E.S.C.

INTRACRANIAL ANEURYSMS

BY

E. G. FEARNSIDES, M.D.Cantab., F.R.C.P.

Assistant Physician to the Hospital for Epilepsy, Paralysis and other Diseases of the Nervous System, Maida Vale; late Medical Registrar to the London Hospital; Beit Memorial Research Fellow

[*Reprinted from* "Brain," Vol. 39, Page 224, *October*, 1916]

LONDON
JOHN BALE, SONS & DANIELSSON, Ltd.
OXFORD HOUSE
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W.

119 Ad/7

95400

4550

22900057631

Med
K34259

WELLCOME INSTITUTE
LIBRARY
welMOmec
WL

34 650 225

INTRACRANIAL ANEURYSMS.

BY E. G. FEARNSIDES, M.D.CANTAB., F.R.C.P.

Assistant Physician to the Hospital for Epilepsy, Paralysis and other Diseases of the Nervous System, Maida Vale; late Medical Registrar to the London Hospital; Beit Memorial Research Fellow.

	PAGE
INTRODUCTION	1
CHAPTER I.—HISTORY	2
CHAPTER II.—THE PATHOLOGY OF INTRACRANIAL ANEURYSMS	8
CHAPTER III.—THE CLINICAL MANIFESTATIONS ASSOCIATED WITH INTRACRANIAL ANEURYSMS OF NON-EMBOLIC ORIGIN	20
CHAPTER IV.—THE CLINICAL MANIFESTATIONS ASSOCIATED WITH INTRACRANIAL ANEURYSMS OF EMBOLIC ORIGIN	53
CHAPTER V.—SUMMARY OF CONCLUSIONS	58
LITERATURE	60
APPENDIX OF CASES	61

THE presence of a saccular aneurysm on one of the basal cerebral arteries at necropsy is one of the commonest pathological findings. Between 1907 and 1913 in the Pathological Institute of the London Hospital 7,924 *post-mortem* examinations of the body were made, in 5,432 of which an examination of the head was permitted, and saccular cerebral aneurysms were discovered in 44 cases. Osler [17] in his first 800 autopsies in Montreal found 12 instances, whilst Newton Pitt [16] in the dead-house of Guy's Hospital between the years 1869 and 1888 found 23 instances in nearly 9,000 examinations. In the past the chief causes of the formation of these aneurysms have been found in (i) embolism and (ii) syphilitic arteritis. In a recent paper entitled "Alterations in Arterial Structure and their Relation to Syphilis," H. M. Turnbull [24] has reviewed our knowledge as to the causations of aneurysms in general and of the morbid changes in the walls of the affected vessels. This paper, which is based upon the findings in the *post-mortem* room of the London Hospital during the years 1907 to 1913 inclusive, proves that the commonly accepted views as to the relation of intracranial aneurysm to an infection with the *Spirochæta pallida* are in need of revision.

CHAPTER I.—HISTORY.

The first definite account which I have been able to find of any intracranial aneurysm was published by Biumi of Milan in 1765, and was reprinted by Sandifort at Leyden in 1778; in this case an aneurysm of the internal carotid artery was found in a female of advanced age who had suffered from several severe rheumatic attacks. In 1814 Blackall in his work on "Dropsy" described an aneurysm of the basilar artery in a young female which had ruptured and produced fatal coma. Hodgson in the following year republished this case in detail and also described a second in which an aneurysm of the anterior cerebral artery, completely filled with blood-clot, was found at the *post-mortem* examination of the brain of an incurable lunatic who had been an in-patient for seventeen years at Bethlem Hospital; he referred to a third case, which had come under his notice, of a ruptured aneurysm of the size of a cherry on one of the cerebral arteries. In 1825 Spurgin of Saffron Walden published a case of aneurysm of the anterior cerebral artery, which occurred in a man of 57 who was suddenly seized with apoplectic symptoms; after a partial recovery and relapse he had a third fit in which he died. The aneurysm was about the size of a hazel-nut and had ruptured. In 1826 Serres published two cases; the first occurred in a man of 59 who died suddenly during convalescence from pneumonia; here, a ruptured aneurysm, of the size of a hen's egg, was found on the basilar artery. The second in a woman aged 53 who had suffered from epileptic seizures and was paralysed on the left side, where a bilocular ruptured aneurysm of the anterior communicating artery was found. Since this time records of cases of intracranial aneurysms are found scattered through medical literature. In 1834 Nebel first collected together and analysed the clinical findings in thirteen cases, and in 1836 Stumpff wrote a graduation thesis on fifteen cases. Brinton [5] in 1852, after describing a case of unruptured aneurysm of the anterior communicating artery, appended a table in which he gave statistical details of forty-nine cases. Gull [11] in 1859 entered more fully into the symptoms caused by aneurysms of the cerebral vessels and endeavoured to establish means by which they might be diagnosed during life, but concluded that "although we may from the circumstances sometimes suspect the presence of aneurysm within the cranium, we have at the best no symptoms upon which to ground more than a probable diagnosis."

The series of papers which Lebert [15] published in 1866 are classical contributions to our knowledge of cerebral aneurysms. Lebert collected the records of 86 cases. He found that 47 of these had occurred on the carotid system of the arteries at the base of the brain and thirty-eight on the vertebral system, and that the incidence had been greatest on the middle cerebral arteries. Fifty-two of his cases had occurred in males and thirty in females; the arteries on the two halves of the body had been affected equally, except in the case of the internal carotid and posterior communicating arteries, where there was a preponderance on the left side. He first noted the frequency of the association of intracranial aneurysm with cardiac disease, but did not trace any causative relationship between these conditions. He gave an extremely lucid account of the clinical manifestations which had been observed in cases of cerebral aneurysm, and stated that in two-thirds of the cases of aneurysm of the middle cerebral arteries, and in 56 per cent. of all cases, rupture of the aneurysm had preceded and led to death. He correlated the common latency and exacerbation of symptoms with the liability of the aneurysms to partial ruptures. As to causes of cerebral aneurysms he considered mechanical, traumatic, and histological factors; in his opinion the majority were due to a "parenchymatous arteritis." "Calcifying atheroma is rarely found in cases of cerebral aneurysm and is not an important cause of their formation." He considered that the few cases which had occurred in patients giving a history of chronic alcoholism or of syphilitic infection did not support the hypothesis that these conditions bear any direct relationship to the formation of cerebral aneurysms. From his researches he was led to believe "that in a number of cases a definite diagnosis of cerebral aneurysm is possible."

Church [8] in 1870 first traced the causative relationship between the formation of intracranial aneurysms and vegetative endocarditis. Church was able to collect 13 cases, four of his own and nine from the literature, of cerebral aneurysms occurring in patients under 20 years of age due to emboli arising from endocarditic vegetations; these occurred on the right middle cerebral artery in 3 cases, on the left middle cerebral artery in 2 cases, on the basilar artery in 2 cases, on the posterior communicating artery in 2 cases, on the posterior cerebral in 1 case, on the right anterior cerebral in 1 case, on the left anterior cerebral in 1 case and on the left internal carotid in 1 case. At the end of his paper he stated that "further research has convinced me that intracranial aneurysms in the young are nearly always due to embolism."

Bartholow [1] in 1872, after reporting one new personally observed and six newly collected cases, again reviewed the literature. From his studies of the records of 114 cases he concluded "there is much reason for believing that syphilis is the chief cause of the formation of intracranial aneurysms" and that after syphilitic aneurysms in order of frequency come those due to embolism from cardiac disease.

Ponfick [21] in 1873 clearly demonstrated the causative relationship between emboli and aneurysmal formation. The detailed description of the organs in his cases proves that all were examples of progressive, ulcerative endocarditis, but in the article of his paper he failed to recognize the infective nature of the emboli and attempted to explain the formation of the aneurysms on a mechanical basis.

Peacock [20] in 1876 analysed the pathological findings in cases of cerebral aneurysm. He reported 3 new cases and collected 86 from literature; he found that the internal carotid arteries had been affected in 12 cases, the anterior cerebral arteries in 4 cases, the anterior communicating arteries in 5 cases, the middle cerebral arteries in 27 cases, the vertebral arteries in 5 cases, the basilar arteries in 22 cases, the cerebellar arteries in three cases, and the posterior cerebral arteries in 6 cases. Death occurred from rupture of the sac in 17 of the 27 cases of aneurysm of the middle cerebral artery and in 14 of the 22 cases of aneurysm of the basilar artery. Of these aneurysms, 40 occurred on the left side and only 31 on the right, and 37 occurred in males and 43 in females. In Peacock's list no case of aneurysm of the posterior communicating artery appears, but, as Peacock notes, Bartholow in 1872 had already reported 8 such aneurysms. As to causation he remarks that "in some cases the disease seems to have orginated in embolism" . . . "not infrequently the aneurysms seem to be caused by syphilis" . . . "the other cerebral arteries are often extensively diseased in cases of aneurysm, being thickened, atheromatous, and indurated, but in some cases the disease of the vessels is said to have been limited to the immediate seat of the aneurysm."

Eppinger [9], whose classical work on the pathology of aneurysms in general appeared in 1887, insisted that in the small and smallest arteries, like those forming the circle of Willis and its branches, many aneurysms have a congenital origin and are due to an inborn defect of the elastic properties of the arterial wall; in others congenital defects with subsequent degenerative or atheromatous processes complicating the defect are the essential factors in causation, whilst in a third group the aneurysms owe their origin to the local action of infective emboli, and in a fourth are due to local non-granulomatous inflammatory changes in the wall of the artery.

Newton Pitt [16], in his third Goulstonian Lecture delivered in 1890, gave an account of 23 cases of cerebral aneurysms which had occurred amongst nearly 9,000 patients who had been examined *post-mortem* at Guy's Hospital between the years 1869 and 1888. His main conclusions were: (1) It is most exceptional to find a cerebral aneurysm which is not associated with fungating endocarditis. (2) The lodging of a septic embolus is the starting point of the changes which take place in the vessel. (3) The clot inflames and in most cases disappears, the vessel dilating from the inflammatory changes in its coats. (4) An aneurysm may form and rupture in three weeks. (5) The onset of the embolism may not be marked by any symptoms, and the rupture of the aneurysm may be the first indication that there is danger.

In 1893 Gowers [10], in his manual of diseases of the nervous system, gave a general account and review of the subject of intracranial aneurysm and discussed the causes, general pathology, symptomatology, course, diagnosis and prognosis. Amongst causes he discussed (1) primary degeneration of the arterial walls, (2) injury, (3) syphilitic disease, (4) embolism. "Primary degeneration is an occasional cause in the second half of life . . . atheroma seldom leads to simple dilatation." With regard to syphilis he stated that "an aneurysm has been frequently met with in cases of constitutional syphilis in young adults, in whom no other cause of aneurysm was discoverable." "Embolism is probably the most frequent cause in the first half of life. There are few cases during this period, without indications of syphilis or injury, in which there is not evidence of past or present heart disease." He gave a list of the numerical frequency in 154 cases obtained by combining the statistics of various writers and found that there had been on the middle cerebral 44 cases, basilar 41, internal carotid 23, anterior cerebral 14, posterior communicating 8, anterior communicating 8, vertebral 7, posterior cerebral 6, and on the inferior cerebellar 3.

Von Hofmann [25] in 1894 published an account of the pathological changes and clinical manifestations which he had observed in 78 new cases of intracranial aneurysm. The order of frequency of affection of the various arteries in his cases was: (1) middle cerebral, (2) internal carotid, (3) anterior communicating, (4) basilar, (5) vertebral, (6) anterior cerebral. He found that the posterior communicating and posterior cerebral arteries had been only seldom affected, and did not find a single instance of a case of aneurysm of the cerebellar arteries. He observed that the favourite sites are (a) the bifurcations of the larger vessels, and (b) the points of division of their branches. He discussed the relation of intracranial aneurysms to atheroma, syphilitic arteritis, embolism, and trauma, and concluded that neither atheroma, syphilis nor trauma are common direct causes of the formation of aneurysms; atheroma and a heightened-blood pressure are important factors in the causation of rupture, and the syphilitic endarteritis of Heubner may exceptionally give rise to aneurysmal formation. Of his cases 70 per cent. were females, and 30 per cent. were males. Fifty-eight were patients brought up to hospital by the police; 8 of these were found dead, 3 in the w.c., 2 in bed, 1 in a cellar, and 1 in a shop, and 8 others died before admission to hospital. He laid great stress upon the importance of looking for intracranial aneurysms in cases suspected of a violent death.

In 1901 Simmonds [23], following up the work of Ponfick and Eppinger, described the minute pathological and bacteriological features which he had found in seven cases of intracranial aneurysm due to infective emboli. All his cases were examples of progressive, malignant endocarditis where the septicæmia was of coccal origin; they occurred in children or young adults who at autopsy showed vegetations on the valvular endocardium and single aneurysms on the cerebral arteries, but no other abnormalities in the cardio-vascular system or changes in the kidneys.

Beadles [2] in 1907, in his Lecture before the Royal College of Surgeons, discussed the symptomatology of aneurysms of the larger cerebral arteries, basing his remarks on the notes of 555 cases of aneurysms of the cerebral arteries found after death, accounts of 441 of which had appeared in the literature and 114 were fresh material. At the end of the lecture he remarks that "notwithstanding the remarks made by some who have specially written on intracranial aneurysms and the writers of some of our leading text-books, the conclusion that I have been forced to draw from a careful study of a large series of cases is, therefore, that it is quite impossible to diagnose an aneurysm of any one of the cerebral arteries except in the most unusual circumstances. Only two or three have ever been diagnosed during life." "The apoplectic symptoms following rupture of an aneurysmal sac are indistinguishable from those of cerebral or meningeal hæmorrhage from other causes. If there is any one sign to which special attention might be drawn, it is the occasional intermittent character of the symptoms. But these form a very small proportion even of the cases where tumour symptoms were present, and, after all, it is not very uncommon for symptoms to show variations in intensity, or even a temporary disappearance, in some other form of tumour and in other cerebral diseases."

The relative importance which, in recent years, authorities have placed upon the different factors which are known to be concerned with the formation of intracranial aneurysms have varied greatly. Thus in a clinical lecture delivered in 1908 John Rose Bradford [4] said: "The formation of a cerebral aneurysm is due to local disease of a vessel; it is a manifestation often of a patchy disease and not of a general arterial disease. . . . The local disease is, in a very large proportion of cases, atheroma, and is atheroma of no doubt syphilitic origin, and we think that in cases of cerebral aneurysms their causation by syphilis is, if anything, a more definite characteristic than it is even in such aneurysms as those of the aorta. In all the cases from which these specimens were removed where a history was obtainable there was no difficulty in tracing the syphilitic infection. . . . So much is that the case that you might make a more general statement and say that in aneurysms involving the larger vessels in young persons the probability of the condition being syphilitic is great. But bear in mind that in the case of the cerebral vessels, great as is the part played by syphilis, there is another factor which does not play any considerable part in the production of aneurysms elsewhere, that is the factor of embolism." Bruns [6], on the other hand, writing in the same year on the pathology of cerebral aneurysms, said that "in the majority of cases the aneurysms are due to arteriosclerosis and atheroma of the intima" and only admitted of syphilis as a cause of these underlying conditions.

Wichern [27] in two recent papers has reported the clinical histories and *post-mortem* findings in 22 new cases and has contributed to the better understanding of the clinical manifestations and to the diagnosis of the condition in patients suffering from aneurysms of the intracranial arteries. According to his analysis of his own cases, 5 were certainly and 6 probably of congenital origin, 5 were embolic, 2 were syphilitic, 2 occurred in patients suffering from general arteriosclerosis, and 2 were "atheromatous," but as far as can be judged from his paper Wichern uses the term "atheroma" to cover changes both in the intima and in the media of the arterial wall. In every one of Wichern's cases the aneurysmal sac had ruptured and in 15 of the 22 clinical or *post-mortem* evidence of previous leakage or rupture, on one or more occasions, was obtained. He insists that aneurysms of cerebral arteries are not rare and that the infrequency of diagnosis in patients is due to the fact that the possibility of cerebral aneurysm as a diagnosis is rarely considered.

CHAPTER II.—THE PATHOLOGY OF INTRACRANIAL ANEURYSMS.

Turnbull [24] divides the chief abnormalities which affect the arteries into: (1) hypertrophies; (2) degenerations; (3) infiltrations; (4) inflammations, and (5) congenital malformations. He confines the term *inflammation* to reactions which are essentially active, and uses the term *degeneration* to distinguish those processes in which degeneration and necrosis are the primary and essential lesions. Only inflammations, degenerations and congenital structural malformations bear any causative relationship to the formation of intracranial aneurysms.

Atheroma is a degeneration which affects and is almost confined to the intima; it is found in both elastic and muscular arteries but is commoner in the large elastic; it is the result and not the cause of the increased blood-pressure which usually accompanies its presence; there is no evidence to be obtained in the *post-mortem* room to show that syphilis induces atheroma.

He finds that medial degenerations have the same cause as atheroma and may assume the forms of (*a*) fatty degeneration; (*b*) calcification associated with fatty degeneration; (*c*) mucous degeneration; (*d*) medial fibrosis.

In inflammations the arteries may be infected from their intima, either by micro-organisms settling upon the surface or by the arrest of infective emboli within their lumen. They may also be infected by micro-organisms reaching the media or adventitia by the vasa vasorum, or by direct inward spread of inflammation from the surrounding tissues. Inflammations of arteries may be divided into the *specific granulomatous inflammations* caused by syphilis, tuberculosis, rheumatism, &c., and *non-granulomatous* where the reaction is caused by the common pyogenic organisms. In his paper he separates for convenience of description the inflammations as they occur in the large elastic arteries, and in the muscular and small elastic arteries, and shows that acquired syphilis is far the commonest cause of aortitis, and is also the commonest cause of inflammation of the other large elastic arteries near the aorta, and, moreover, that syphilitic aortitis is the lesion of acquired syphilis which is found most frequently in the *post-mortem* room of a General Hospital. In the small muscular and elastic arteries, the group to which the cerebral arteries belong, syphilis induces a relatively chronic arteritis, usually spoken of as endarteritis obliterans, in which changes in the intima are conspicuous and produce almost complete occlusion of the lumen. In the cerebral arteries, especially those of the base, arteritis consequent upon syphilis causes the artery to appear to naked-eye examination fusiform, white and thick, to feel firm and solid, and on transverse section to show a cut surface of firm, grey tissue, in the centre of which a minute red lumen may be visible. Microscopically, endarteritis obliterans is characterized by the appearance of an intimal thickening internal to the elastic lamina and the boundary stripe. In the earliest stages round cells are seen between the stripe and the endothelium. Spindle fibroblasts are found later and give rise to the formation of a definite granulation tissue. The granulation tissue ultimately becomes fibrotic and delicate elastic fibrils make their appearance. The elastic lamella and stripe remain unaltered throughout the process; this enables the condition to be recognized easily in sections stained by a specific method for demonstrating elastic tissue and to be distinguished readily from medial and intimal degeneration. The inflammation of the media and adventitia which accompanies the endarteritis conforms in type to the inflammation caused in other tissues by the particular micro-organism.

Turnbull discounts the use of the term *arterio-sclerosis* because it has been applied indiscriminately to hypertrophied arteries, to arteries showing atheromatous or (and) medial degeneration, to arteries infiltrated with amyloid, and to arteries reacting by inflammation. The term means merely "arterial hardening" and does not describe the pathological processes at work.

The term aneurysm (ἀνευρύνω, widen or dilate) is used to describe any dilatation of an artery. In this paper, as in Turnbull's, however, we shall deal only with focal, true, fusiform or saccular aneurysms of a size large enough to be readily visible to the naked eye. *True aneurysms* are dilatations which are bounded by the coats of an artery. *False aneurysms*, on the other hand, are cavities which communicate with the lumen of an artery but are bounded by tissues external to the wall of the artery. True aneurysms frequently rupture and give rise secondarily to false aneurysms; the resulting structure is spoken of as a *mixed aneurysm*. In this paper, all false aneurysms in which there is evidence of a primary dilatation, that is to say, all mixed aneurysms, will be referred to as true. The term "direct rupture" will be employed to denote a rupture which has not been preceded by aneurysm. False aneurysms will, therefore, be included among "direct ruptures."

According to Turnbull, true aneurysms thus described may be divided pathologically into two main groups: (1) *congenital*; (2) *acquired*. In the formation of acquired aneurysms generally, trauma, peptic digestion, inflammations, both granulomatous and non-granulomatous, degenerations, loss of mechanical support and heightened blood-pressure are the only known factors at work. Turnbull has not been able to find, either in the *post-mortem* room or from a study of the literature, any unequivocal evidence that aneurysms of the cerebral arteries are ever due to trauma; digestion by peptic ferments introduced from without and loss of mechanical support also cannot be assigned as important factors in the formation of intracranial aneurysms. The only factors which remain to be considered, therefore, are: (1) congenital; (2) inflammatory, and (3) degenerative.

In the series of cases which Turnbull and I have studied and which form the basis of this paper, no single example of granulomatous inflammation giving rise to a cerebral aneurysm occurred. Such cases, however, in the literature are not rare. They have been found most commonly on the vertebral and basilar system of the basal arteries and descriptions of their morbid characteristics have been given by many authors; Rose Bradford [3], for example, in 1894, showed a good example which occurred on the basilar artery in a patient who at autopsy also showed multiple gummata of the liver. Wichern [28] described two cases (Cases 19 and 20) of ruptured aneurysm of the basilar artery accompanied by diffuse syphilitic endarteritis obliterans of the cerebral vessels; the first was found in a widow of 56, who at autopsy also showed periostitis of the tibia and syphilitic mesaortitis, and the second in a woman of 51.

Unfortunately, as in the majority of my own cases, Wichern's patients were observed some years ago and before the recent development and application of the Wassermann technique, and so far I have not been able to find any more recently described case where the Wassermann reaction was tested both in the serum and in the cerebrospinal fluid *ante mortem* and where at autopsy an aneurysm of one of the cerebral vessels was discovered. In 1913, Langbein [14] published a description of a single case in which a positive Wassermann reaction in both the serum and in the cerebrospinal fluid was found in a man aged 40, which clinically simulated and which he himself recorded as one of recurrent leakages on the part of a cerebral aneurysm; but the patient recovered and the nature of the meningitic affection and the cause of the recurring apoplectic attacks remain extremely doubtful, as no autopsy has as yet been performed.

The relatively high incidence of syphilitic aneurysm of the basilar artery is probably due to the fact that this artery is a large one and that in the smaller cerebral arteries syphilitic inflammation usually leads to such a severe degree of intimal hypertrophy that the lumen of the artery is blocked and thus prevents aneurysmal dilatation. When an extremely intense arteritis, whether granulomatous or non-granulomatous, affects the cerebral vessels, direct rupture or the formation of a false aneurysm may take place.

Aneurysms of the muscular and small elastic arteries frequently result from infection by bacteria which cause acute inflammations. The arteries are most commonly infected by impaction of emboli in their lumina. Aneurysms due to infective embolism are found most frequently in the cerebral arteries; the superior mesenteric artery is the second site of election. In our records they have occurred most frequently in cases of progressive malignant endocarditis. No aneurysms of cerebral vessels were caused by infection from without.

Saccular aneurysms due to medial degeneration, independent of inflammation, are found much more commonly on the cerebral arteries than on any other muscular arteries. In none of the cases of cerebral aneurysm which we have attributed to this class was there evidence at necropsy of active or healed endocarditis. In some there was evidence of cardio-vascular hypertrophy, accompanied by a considerable degree of general arterial degeneration, and in such cases, as Turnbull remarks, there would appear to be an adequate explanation of the formation of the aneurysms. In many of the cases, however, there was no evidence of excessive blood-pressure, either at autopsy or from clinical observations, and the general arterial degeneration and the degeneration of the cerebral arteries were no greater than in the average subject of the same age. In this respect many of the cases of aneurysm contrast sharply with the cases of direct rupture of central cerebral arteries. Direct rupture is usually associated with evidence of much more excessive blood-pressure (hyperpiesia) and arterial degeneration. There appears, therefore, to be an additional factor in the formation of cerebral aneurysms. This factor is, probably, (as Eppinger [9] first suggested) an inherent, inborn weakness due to a congenital abnormality in the structure of the arteries at their points of junction and bifurcation. This conclusion is strengthened by the finding in the *post-mortem* room of the London Hospital in the year following the cases analysed in this paper of an aneurysm at the junction of the left anterior cerebral with the anterior communicating artery, in a child aged 1 year and 7 months, who died of broncho-pneumonia complicating acute gastro-enteritis.

The arteries of the brain all receive their blood from the internal carotid and vertebral arteries, whose anastomoses at the base of the brain form the arterial circle of Willis. On the ventral surface of the fore end of the medulla oblongata the two vertebral arteries unite to form the unpaired basilar artery; this artery courses over the pons and divides anteriorly into the two posterior cerebral arteries which run out almost at right angles to the parent vessel. The internal carotid arteries immediately after reaching the cranial cavity send back the posterior communicating arteries which unite the posterior cerebrals with the carotid system. On the side of the optic tract the internal carotid arteries give off the anterior cerebral arteries, which supply the frontal lobes and corpus callosum of the brain, and continue as the the middle cerebral arteries. The two anterior cerebral arteries are united by the unpaired anterior communicating artery.

The circle of Willis is thus formed by:—

$$\text{basilar artery}\left\{\begin{array}{l}\text{posterior cerebral—posterior communicating—}\\ \text{posterior cerebral—posterior communicating—}\end{array}\right.$$

$$\left.\begin{array}{l}\text{—carotid—anterior cerebral}\\ \text{—carotid—anterior cerebral}\end{array}\right\}\text{anterior communicating.}$$

Although this is the "normal" arrangement, variations are extremely common. Amongst 200 successive observations on the conformation of the circle of Willis in dissecting-room subjects Windle [29] found that the number of arteries and their arrangement was "normal" in 119 (59·5 per cent.); the circle was symmetrical in 76, and symmetrical but for some inequality in size of the posterior communicating arteries in 43 others. In 159 the anterior communicating artery was normal; in 14 cases two anterior communicating arteries were present, in 1 case three were found, in 6 cases the artery was incompletely duplicated, in 6 cases the anterior cerebral arteries were united at their origin, in 2 cases both an union of the anterior cerebral arteries at their origin and also an ordinary anterior communicating artery were found, and in 9 cases a mesial artery arose from the anterior communicating artery. In 181 the anterior cerebral arteries were normal; in 9 cases two anterior cerebral arteries were discovered with in addition a mesial artery arising from the anterior communicating artery; in 8 cases the anterior cerebrals were united for a distance and in 3 cases only a single mesial anterior cerebral artery was found. In 173 the posterior cerebral arteries and in 175 the posterior communicating arteries were normal. In size the posterior communicating arteries were often unequal, the right was larger than the left in 28 cases, and the left larger than the right in 15; both were small in 7 cases and both were absent in 3 cases; in 9 the right was absent and in 13 no left posterior communicating artery could be found. Wyrubow [30] amongst 112 successive examinations of the conformation of the circle of Willis in patients who had died from mental disease found that 22·3 per cent. showed abnormalities of the anterior cerebral arteries, and Parnisetti [19] at the autopsies on 87 criminals found only 51 (58·62 per cent.) with normal arrangements of the circle of Willis. Rothmann [22] states that in apes and anthropoids the anterior communicating artery is usually absent and the circle of Willis is thus not completed. Hofmann [13] found that the circle is usually incomplete anteriorly in frogs, salamanders and birds, and showed that amongst the various sub-groups of the mammalia the arrangements vary: zoologically closely related mammalia often show important differences in the arrangement of the basal cerebral arteries. Thus amongst the Ungulata, in the pig the circle is usually completed anteriorly; in the cow, on the other hand, this is not so. Amongst the Anthropoidea, in the gorilla no anterior communicating artery is found; in man this artery is normally present.

From these observations we see that morphological variations of a gross character are extremely common in the arteries forming the circle of Willis in all classes of the Vertebrata, and microscopical investigations have shown that minor changes in the elastic layer of the media of the arteries are also common in the cerebral arteries of patients who show no gross saccular aneurysmal dilatations. Most of the variations, whether major or minor, are found at the points of junction or of bifurcation of the vessels, and it is at these points that we find aneurysms visible to the naked eye of supposedly congenital origin.

In no case in this series of cases could any relation to trauma as a possible cause of the formation of intracranial aneurysm be traced.

Occurrence.—During the years 1907 to 1913 at the Pathological Institute of the London Hospital 7,924 *post-mortem* examinations of the body were made, in 5,432 of which an examination of the head was permitted. In these cases the following aneurysms were discovered:—

True Aneurysms of Aorta—	
Due to syphilitic inflammation	175
,, tuberculous inflammation	1
,, non-granulomatous inflammations	4
,, atheromatous degeneration	4
	184
Dissecting Aneurysms of Aorta—	
Due to non-granulomatous inflammation	5
,, developmental deficiency	2
	7
True Aneurysms of Other Large Elastic Arteries—	
Due to syphilitic inflammation	43
True Aneurysms of Muscular and Small Elastic Arteries (excluding the Cerebral Arteries)—	
Due to syphilitic inflammation	6
,, tuberculous inflammation (probably not all reported, most occur with pulmonary vomicæ)	10
,, infective embolism	18
,, spread of inflammation from without	3
,, medial degeneration	7
	44
True Aneurysms of Cerebral Arteries—	
Due to infective embolism	15
,, medial degeneration	36
	51

From this table we see that aneurysms of the aorta occurred in 191 cases or in 2·4 per cent. of examinations of the body, that 51 aneurysms of the cerebral arteries occurred in 44 cases or in 0·80 per cent. of examinations of the head, and that 44 aneurysms of the other large elastic arteries also occurred, i.e. one in 0·54 per cent. of examinations of the body. These figures contrast strongly with those from Crisp which Osler [17] quotes in his "System of Medicine," where amongst 501 aneurysms, 184 occurred in the aorta, 137 in the popliteal artery and only 7 intracranial aneurysms were discovered.

The commonest cause of all aneurysms is syphilitic inflammation of the wall of an artery. Ninety-six per cent. (96·2 per cent.) of the aneurysms of the aorta, 100 per cent. of the aneurysms of the other large elastic arteries, but only 6·25 per cent. of the aneurysms of the muscular and small elastic arteries, to which class the arteries of the circle of Willis and the cerebral arteries generally belong, were consequent on the local weakening of the arterial wall due to the inflammatory reaction consequent on the activity of the *Spirochæta pallida*, and in no case in this series of necropsies was an intracranial aneurysm due to this cause found; at the same time the low rate of incidence of aneurysms on the vertebral system of arteries in this series of cases, compared with those recorded by others, is extremely noteworthy.

In this series of examinations of the brain 31 cases showed thirty-six aneurysms of the cerebral arteries due to non-inflammatory medial change, and 13 cases showed fifteen cerebral aneurysms caused by infective embolism. Aneurysms due to non-inflammatory medial change occurred therefore in 0·55 per cent. of examinations of the brain, and aneurysms due to inflammatory changes in the cerebral arteries associated with infective embolism in 0·24 per cent.

Of these aneurysms on the cerebral arteries, death had been caused by rupture in 25 cases where the aneurysm was due to non-inflammatory changes, and in 10 cases where the aneurysm had resulted from infective embolism, or together in 4·42 per mille of the autopsies at the London Hospital during the years 1907 to 1913. During the same period intracranial hæmorrhage due to the direct rupture of central cerebral arteries consequent on medial degeneration was found in 72 cases or in 9·08 per mille of the autopsies, giving the relative frequency of cerebral hæmorrhages due to direct rupture compared with those following true aneurysmal formation of 35 to 72, or 1 to 2·05.

The following is a list of the sites of the cerebral aneurysms classified according to their pathological causes :—

	Embolic	Medial degeneration
Left intracranial carotid	1	1
Origin of cortical branch from left carotid	0	1
Junction right carotid with posterior communicans	0	1
Junction left carotid with posterior communicans	0	2
Junction right carotid with middle cerebral	0	2
Right middle cerebral near base of brain	1	8
Left middle cerebral near base of brain	1	2
Cortical branches of right middle cerebral	4	2
Central branches of right middle cerebral	0	1
Cortical branches of left middle cerebral	3	0
Central branches of left middle cerebral	1	0
Junction of right anterior cerebral with anterior communicans	0	2
Junction of left anterior cerebral with anterior communicans	0	7
Right anterior cerebral	2	2
Left anterior cerebral	1	1
Left anterior communicans	0	1
Junction of right posterior cerebral and posterior communicans	0	1
Bifurcation of basilar	1	0
Left posterior cerebellar branch of basilar	0	1
Left anterior inferior cerebellar	0	1
	15	36

Multiple cerebral aneurysms due to medial change were found in four of the thirty-one cases:—

Case 3 showed two aneurysms on central branches of the right middle cerebral artery, one of which had ruptured.

Case 4 exhibited two intracranial aneurysms, one of which at the junction of the left anterior cerebral and the anterior communicating arteries had ruptured, and the other of which at the junction of the right carotid and posterior communicating arteries was unruptured.

Case 11 showed two aneurysms, neither of which had ruptured; one of these was found on the right middle cerebral artery near the base of the brain, and the other at the junction of the left anterior cerebral and anterior communicating arteries.

Whilst Case 26 showed three aneurysms; one occurred at the junction of the left intracranial carotid and posterior communicating arteries and was ruptured, and the other two were found on the right middle cerebral artery and had not ruptured. Thus amongst 31 cases showing intracranial aneurysms, due to medial change, 4 cases exhibited nine aneurysms, and 31 cases thirty-six aneurysms.

Fifteen saccular aneurysms due to medial degeneration occurred at the actual point of junction of the arteries of the circle of Willis (Cases 4 (three examples), 5, 6, 8, 10, 11, 16, 17, 23, 24, 26, 27, 30). In addition, six were found close to such junctions and ten arose from the point of origin of branches. In two cases no note was made upon the relation to points of junction or bifurcation.

In one case two minute aneurysms were situated upon central cerebral branches. Of the remaining 34 due to medial degeneration, all except one sprang from cerebral arteries of the first order. Only three were connected with the posterior part of the circle of Willis.

The various sites of the intracranial aneurysms caused by infective embolism which occurred during the years 1907 to 1913 are given in the table on next page.

In this table are shown nineteen aneurysms of the muscular and small elastic arteries which occurred in 13 cases. Amongst these cases were found three aneurysms on visceral arteries and one on the profunda femoris artery. During the same period in cases where an examination of the head was permitted and no cerebral aneurysms were found, there occurred two aneurysms on the superior mesenteric artery, two on the splenic artery and one on the left brachial artery, giving the proportion of aneurysms due to infective embolism amongst 5,324 examinations of fifteen cerebral to nine on other arteries. In the 13 cases showing at autopsy cerebral aneurysms, ten of the cerebral

Number of cases	Right middle cerebral	Left middle cerebral	Branch on vertex right middle cerebral	Branch on vertex left middle cerebral	Right anterior cerebral	Left anterior cerebral	Left intra-cranial carotid	Bifurcation of basilar	Superior mesenteric	Cœliae axis	Left renal artery	Left profunda femoris	—
4	R 4R R	—	—	—	—	—	—	—	—	—	—	—	I, IV, VIII, X.
1	—	—	—	—	1R	—	—	—	—	—	—	—	II.
1	—	—	—	—	—	1	—	—	—	—	—	—	VI.
1	—	—	1R	—	—	—	—	—	—	—	—	—	VII.
1	—	1R	—	—	—	—	—	—	—	—	—	—	III.
1	—	1R	—	1	—	—	—	—	—	—	—	1R	V.
1	—	—	—	—	—	—	1	—	—	1R	1	—	IX.
1	—	—	—	—	—	—	—	1	—	—	—	—	XI.
1	—	1R	—	—	—	—	—	—	1	—	—	—	XII.
1	—	1R	—	—	1R	—	—	—	—	—	—	—	XIII.
13	4	4	1	1	2	1	1	1	1	1	1	1	—
—	3R	4R	1R	—	2R	—	—	—	—	—	—	—	—

aneurysms and two of the other aneurysms had ruptured and given rise to considerable hæmorrhage. *Post-mortem* evidence of progressive malignant endocarditis was found in 10 of the cases; in 1 case a pyæmia subsequent to a retropharyngeal abscess, and in 1 case a pyæmia consequent on puerperal sepsis were found, and in 1 case the primary seat of the infection was not ascertained.

Multiple aneurysms due to emboli in cases of general septicæmia occurred in two cases.

Case V showed a ruptured aneurysm on a perforating branch of the left middle cerebral artery and an unruptured aneurysm on a cortical branch of the same artery; whilst Case XIII showed two ruptured cerebral aneurysms, one on a cortical branch of the left middle cerebral artery and a second on a cortical branch of the right anterior cerebral artery.

In 3 cases aneurysms on arteries in other parts of the body were also found.

Case V showed a ruptured aneurysm on the left profunda femoris artery; Case IX showed a ruptured aneurysm on the cœliac axis artery and an unruptured aneurysmal dilatation of the left renal artery; and Case XII an unruptured aneurysm on the superior mesenteric artery. Thus, of 13 cases 2 showed multiple intracranial aneurysms and 3 showed aneurysms of vessels in other parts of the body.

In this series of cases 17 intracranial aneurysms due to medial changes occurred on the left side, 19 on the right, whilst of those caused by infective embolism 7 were found on the left side and 7 on the right and 1 occurred at the bifurcation of the mesial basilar artery. Thus 24 occurred on the left half of the body and 26 on the right.

SIZE—

(*a*) *Of Cerebral Aneurysms due to Medial Degeneration.*

Smaller than a pin's head	Case 11	1
Size of a pin's head	Cases 10, 11, 21, 28	4
Size of half-pea	Case 3	1
Size of a grape-stone	Case 26 (two examples)	2
Larger than a grape-stone	Case 26	1
Size of a pea	Cases 1, 3, 4, 4, 5, 6, 7, 12, 16, 20, 27, 29	12
Size of a cherry-stone	Cases 13, 15, 25	3
Size of a currant grape	Case 23	1
Size of a haricot-bean	Case 31	1
2 mm. in diameter	Case 2	1
3 mm. in diameter	Case 19	1
6 mm. in diameter	Cases 8, 17, 18	3
14 mm. in diameter	Case 24	1
15 mm. by 10 mm.	Case 30	1
30 mm. in diameter	Case 14	1
No notes on size	Cases 9, 22	2
		36

(b) Of Cerebral Aneurysms due to Infective Embolism.

Size of a pea	Cases II, V, VI	3
Size of a cherry-stone	Cases IV, VIII, X	3
Size of two peas	Case I	1
Size of three peas	Case III	1
Size of a cherry	Case VII	1
"Dilatation of vessel"	Cases V, IX, XI	3
25 mm. in diameter	Case XII	1
No notes on size	Case XIII (two examples)	2
		15

Sex.—Cerebral aneurysms due to medial degeneration were found in 18 males and in 13 females, and due to infective emboli in 8 males and 5 females, giving a proportion in the whole series of cases of 26 males to 18 females.

Age.—If we divide up our cases of saccular aneurysms of cerebral arteries due to changes in the media according to the degree of cardio-vascular hypertrophy we find that:—

In two patients, aged 51 and 48, there was *very great* cardio-vascular hypertrophy.

In four patients, whose ages varied from 60 to 44, and averaged 50, there was *great* cardio-vascular hypertrophy.

In three patients, whose ages were 86, 75 and 50, there was *considerable* cardio-vascular hypertrophy.

In three patients, aged 55, 53 and 46, there was *moderate* cardio-vascular hypertrophy.

In three patients, aged 72, 67 and 60, there was *slight* cardio-vascular hypertrophy.

In fifteen patients, whose ages varied from 53 to 19 and averaged 38, there was *no* cardio-vascular hypertrophy.

And these cases were distributed between	50 and 54	2
,, ,, ,, ,,	45 ,, 49	3
,, ,, ,, ,,	40 ,, 44	4
,, ,, ,, ,,	35 ,, 39	2
,, ,, ,, ,,	30 ,, 34	0
,, ,, ,, ,,	25 ,, 29	1
,, ,, ,, ,,	20 ,, 24	2
,, ,, ,, ,,	15 ,, 19	1

and in one case only an examination of the head was permitted.

A study of the incidence, therefore, in these thirty-one cases shows:—

That between the ages of	85 and 89	there occurred	1 case
,, ,,	80 ,, 84	,,	0 ,,
,, ,,	75 ,, 79	,,	1 ,,
,, ,,	70 ,, 74	,,	1 ,,
,, ,,	65 ,, 69	,,	1 ,,
,, ,,	60 ,, 64	,,	2 cases
,, ,,	55 ,, 59	,,	0 ,,
,, ,,	50 ,, 54	,,	6 ,,
,, ,,	45 ,, 49	,,	7 ,,
,, ,,	40 ,, 44	,,	6 ,,
,, ,,	35 ,, 39	,,	2 ,,
,, ,,	30 ,, 34	,,	0 ,,
,, ,,	25 ,, 29	,,	1 case
,, ,,	20 ,, 24	,,	2 cases
,, ,,	15 ,, 19	,,	1 case
			31

The age-incidence of cerebral aneurysms of embolic origin is determined by that of chronic septicæmic and pyæmic conditions. In this series of cases

Between the ages of	5 and 9	there occurred	1 case
,, ,,	10 ,, 14	,, ...	0 ,,
,, ,,	15 ,, 19	,, ...	4 cases
,, ,,	20 ,, 24	,, ...	3 ,,
,, ,,	25 ,, 29	,, ...	0 ,,
,, ,,	30 ,, 34	,, ...	1 case
,, ,,	35 ,, 39	,, ...	2 cases
,, ,,	40 ,, 44	,, ...	2 ,,
			13

Chapter III.—The Clinical Manifestations associated with Intracranial Aneurysms of Non-Embolic Origin.

The course of illness and the actual manifestations of departure from healthy states which occur in patients who at autopsy exhibit aneurysmal dilatation *with rupture* of the larger cerebral arteries form a fairly definite nosological group. Nevertheless these deviations from the normal so closely resemble those seen in morbid intracranial conditions owning other causes that Gull's statement written in 1859 still holds true "that although we may from the circumstances sometimes suspect the presence of an intracranial aneurysm, we have at the best no symptoms upon which to ground more than a probable diagnosis.

In nervous tissue, as contrasted with the other tissues of the body, function is more strictly localized. The interferences with function which result from the appearance of morbid changes in the vessels supplying the central nervous system depend upon the particular portions of the nervous axis which are affected secondarily. The finding on a given date of no single set of clinical manifestations of itself can enable us to pronounce a definite opinion upon the nature of the underlying pathological processes; tumour, syphilitic, and other forms of meningitis, aneurysm, &c., occurring at the same point, give rise to identical interferences, but for each the course of the disease is more or less characteristic, and if the possibility of aneurysm as a diagnosis were more frequently considered a larger number of cases would be recognized during life. In a considerable proportion of the cases which are included in this series a shrewd guess as to the causative factor was made before autopsy, and in a still larger number such a guess ought to have been hazarded. In the *post-mortem* room an aneurysm of one of the cerebral arteries is a relatively common finding, and yet in the past when considering at the bedside the causative possibilities in a given patient cerebral aneurysms have rarely been suggested.

In 80·7 per cent. of the cases (25 out of 31) of non-embolic aneurysms due to changes in the media included in this series, signs attributable to cerebral hæmorrhage or to a local affection of the meninges consequent upon the *rupture* of the sac occurred, whilst in 16·1 per cent. (5 out of 31) an unruptured cerebral aneurysm, which, so far as is known, had not led during life to any definite manifestations, was found, and in 3·2 per cent. (1 case out of 31) in the history given by the patient on his admission to hospital was an account of a seizure and of an "influenzal" attack followed by much frontal headache and deafness of the right ear, which might or might not have been due to the development of an aneurysm at the junction of the right carotid and middle cerebral arteries.

Cerebral aneurysms which do *not* rupture rarely give rise to symptoms.

Death occurred in Case 7 from broncho-pneumonia consequent upon septic pharyngitis with abscess formation in the neck in a man aged 86, who five weeks previously awakened one morning complaining of an inability to utter words. At the autopsy atheroma of most of the cerebral arteries and unruptured aneurysm, of the size of a pea, was found on the right middle cerebral artery near the base of the brain. In Case 8 the patient, a man aged 75, died forty hours after a suprapubic prostatectomy and an unsuspected and unruptured aneurysm, 6 mm. in diameter, was found at the junction of the left anterior cerebral and anterior communicating arteries. Case 11, a fish-curer, aged 53, for two and a half years before death suffered from hæmaturia and renal insufficiency associated with congenital cystic kidneys. He died "uræmic," and at autopsy two small unruptured aneurysms were found, one at the junction of the left anterior cerebral and anterior communicating arteries, of the size of a pin's head, and the other, rather smaller, on the right middle cerebral artery near the base of the brain.

Case 19 was that of a married woman aged 48; for five years she had suffered from symptoms attributable to cholelithiasis, and eighteen days before death was admitted to hospital with an acute surgical condition of the abdomen. Laparotomy was immediately performed; a tumour of the gall-bladder was removed and a subphrenic abscess drained. After the operation her general condition grew worse, hæmatemesis from acute ulceration of the stomach set in and she died of internal hæmorrhage. At the autopsy an unruptured aneurysm on the right middle cerebral artery, 3 mm. in diameter, was found near the base of the brain. Case 21 was admitted to hospital with symptoms of Korsakow's psychosis and alcoholic peripheral neuritis, from which she died. At autopsy cirrhosis of the liver, fibrosis of the pancreas and a certain degree of cirrhosis of the kidneys, and in addition an unruptured aneurysm, of the size of a pin's head, on the left middle cerebral artery about 1 in. from its origin, were found.

Case 20 is our only example of a patient in whom an unruptured aneurysm at the time of its development probably caused certain intracranial disturbances which came on acutely and then remained quiescent until the death of the patient from an intercurrent disease bearing no direct relation to the vascular system. This man, when aged 47, was admitted to hospital with signs of acute diffuse myelitis from which he died. He gave the history that six years before death he had suffered a slight seizure followed by general weakness, irritability and depression, and that about a year previously he had had an attack of "influenza" followed by deafness of the right ear and much frontal headache. Here an unruptured aneurysm, of the size of a large pea, was found at the junction of the right carotid and middle cerebral arteries, in addition to diffuse inflammation of the spinal cord.

Two of the patients included in this series *died before admission to hospital.* Of the previous history of Case 1 nothing is known. The patient, a man aged about 51, was found by the police lying dead in the street; a ruptured aneurysm of the size of a pea was found on the right middle cerebral artery near the base of the brain. The case was one of hyperpiesia; atheroma was present in the wall of the aneurysm, in most of the cerebral arteries and in most of the systemic arteries, and the heart showed left-sided hypertrophy. Case 28 was that of a clerk, aged 22, who had gained some local fame as a cyclist; after riding a mile at a public cycle-race meeting he "came over giddy," got off his machine, lay down on the side of the track, lost consciousness almost immediately and died within half an hour of the onset of the symptoms. At the autopsy a ruptured aneurysm on the right middle cerebral artery, 4 cm. from its origin and near the base of the brain, of the size of a pin's head, was discovered; in the rest of the cardiovascular system no gross changes were found.

Ten cases were admitted *after a first apoplectic seizure, and died without ever recovering consciousness.* Case 9, a man aged about 50, lived only one hour and a half after his admission; in this case the diagnosis of "fractured base" was made owing to the history of a fall in the street and the finding on examination of a bruise on the occiput. At the *post-mortem* examination a ruptured aneurysm was found on the right anterior cerebral artery just beyond the origin of the anterior communicating artery: and in the great longitudinal fissure old altered blood-clot and staining of the meninges was present, suggesting that a partial rupture had occurred on some previous occasion.

Case 10 was that of a greengrocer, aged 55; he, too, was found lying in the street by the police and died in coma about an hour after admission. This patient showed left hemiplegia, unequal fixed pupils—the right larger than the left—hyperpiesia and enlargement of the left ventricle. At the autopsy a ruptured aneurysm of the size of a pin's head was found at the junction of the right anterior cerebral and anterior communicating arteries.

Case 24 died unconscious within a few hours after admission. The patient was a woman hawker, aged 40, who was found by the police lying stertorous and deeply cyanosed on the roadside. On admission, both pupils were widely dilated, and neither reacted to light, and both plantar reflexes gave an extensor response. Here the ruptured aneurysm, 1·4 cm. in diameter, was found at the junction of the right posterior cerebral and posterior communicating arteries.

Case 18 was that of a married woman, aged 49, who was said to have suffered for four months from nocturnal frequency of micturition, occasional attacks of headache, and a general nervous state of irritability attributed to the onset of the menopause. Eight days before death an extremely severe attack of headache leading to nausea and vomiting was complained of; the headache persisted for six days, and then the patient became delirious and lost consciousness. On admission ten hours before death she was deeply unconscious, and no local signs of interference with motion or with the functions of the cranial nerves were discovered. Two hours after admission she exhibited three generalized convulsions, developed hyperpyrexia (T. = 106° F.), and died. The aneurysm, which had ruptured, was found on the left anterior communicating artery, and was 6 mm. in diameter; there were few signs of cardio-vascular disease.

Case 27, a compositor, single, aged 28, had suffered for eighteen months from attacks of left-sided headache, accompanied during the last six months by severe nausea and giddiness, and usually some vomiting; sixteen hours before death he got up in the morning feeling particularly well, and went to the bathroom, where he fainted and lost consciousness. Fourteen hours after this seizure he was admitted moribund, and within ninety minutes died with increasing coma and cyanosis. An aneurysm, of the size of a pea, which had ruptured, was found on the left intracranial carotid artery at the origin of a branch to the temporal pole. The rest of the cardio-vascular system showed few signs of disease.

Case 17 was that of a labourer, aged 51, who had been addicted to alcoholic excess, and for several winters subject to attacks of "winter-cough." About a month before death manifestations of cardiac failure appeared without any symptoms referable to the nervous system. Eighteen hours before death he was seized with a sudden intense headache, leading to vomiting and the appearance of a right-sided extensor plantar response; he lost consciousness, and died eighteen hours later. A ruptured aneurysm, 6 mm. in diameter, was found at the *post-mortem* examination on the left anterior communicating artery.

Case 13, an old woman, aged 72, who lived alone, was found lying unconscious in her kitchen; she died twenty-eight hours later. On admission neither pupil reacted to light, and both plantar reflexes gave an extensor response. An aneurysm, of the size of a cherry-stone, which had burst, was found on the left anterior cerebral artery just beyond the origin of the left anterior communicating artery. In this case there was moderate general arterial degeneration and moderate cardio-vascular hypertrophy.

A married woman, aged 48 (Case 2), was suddenly seized with apoplexy whilst in the street, and died unconscious thirty-six hours later. In this case widespread cardio-vascular changes were found at the autopsy, together with a ruptured aneurysm, 2 mm. in diameter, on the right middle cerebral artery near the base of the brain.

Case 5 was that of a man, aged 49, a painter by trade, who, whilst at work in the hospital garden, fell, and was found later lying unconscious. He was admitted immediately, but never recovered consciousness; he died on the third day after the seizure of respiratory failure. Lumbar puncture in this case showed the presence of much free blood in the cerebrospinal fluid, and the examination of the patient revealed general vascular disease, hyperpiesia, right facial paresis, and evidence of bilateral corticospinal interference. At the autopsy atheroma of most of the cerebral vessels, general arterial degeneration and hypertrophy of the left ventricle were found, and also a ruptured aneurysm of the size of a pea at the junction of the left anterior cerebral and anterior communicating arteries.

In Case 6 the patient, a married woman, aged 44, had suffered for several years from "ill-health, depression, and hysterical fits"; for these she took alcohol to excess. Twenty-four days before death she began to complain of severe pain in the head, acute diarrhœa, and much vomiting; this attack lasted three days. Then she improved somewhat, but remained drowsy, and talked irrationally. Fourteen days before death she was admitted to hospital in a drowsy, apathetic, lethargic state, passing urine and fæces under her. On examination, the calves were exquisitely tender to pressure, the knee-jerks and ankle-jerks were abolished, but the left plantar response was extensor; from time to time the patient showed attacks of irregular movements of the right upper extremity. In this state she remained for several days, then signs of broncho-pneumonia developed, and she died. The autopsy showed a ruptured aneurysm of the size of a pea at the junction of the left anterior cerebral and anterior communicating arteries, whilst both anterior cerebral arteries arose from the left internal carotid. All the cerebral and most of the systemic arteries showed medial and atheromatous changes, and the left ventricle was much enlarged.

The clinical history in this case, although the patient never regained complete consciousness after her first seizure, forms a connecting link with the more definite and more easily recognized group of cases in which multiple seizures at varying intervals and signs of localized interferences at the base of the brain precede the fatal issue.

When death in a case of ruptured cerebral aneurysm follows directly upon the first rupture of the sac, the differential diagnosis between intracerebral and intrameningeal hæmorrhage can only be made after a full consideration of all the attendant *localizing* symptoms and signs; and these often are insufficient to lead to the formation of a correct opinion. In 41·9 per cent. of cases (13 out of 31) of cerebral aneurysms of non-embolic origin in this series, however, an especial feature, which, so far as my experience goes, amongst all other intracranial conditions is characteristic of ruptures of this class of aneurysms, was also present, viz.: an account in the history of the patient's manifestations of a sudden exacerbation, with apoplectiform onset, of symptoms and signs followed by a period of varying, but often considerable, duration, in which no fresh developments appeared, followed later by a second and a third, and even by a fourth, seizure, in the last of which death from respiratory failure and medullary pressure occurred, and correlated with this the finding at autopsy of evidence of leakages of different ages owing to partial ruptures of the aneurysmal sac.

Wichern [27 and 28], from a study of the literature of cerebral aneurysms in general, found that multiple seizures had occurred in 57 out of 183 cases (31·1 per cent.), figures which correspond well with the 13 out of 44 cases (29·6 per cent.) of all classes of cerebral aneurysms which, on the one hand, manifested multiple seizures, and, on the other, were examined in the *post-mortem* room of the London Hospital during the period under consideration.

A case of aneurysm at the junction of the left internal carotid and posterior communicating arteries of congenital origin which first give rise to symptoms at the age of 19. *Three partial ruptures giving rise to apoplectiform attacks. Admitted to hospital showing complete paralysis of the left third cranial nerve, optic neuritis on left and proptosis on right, and left cerebellar posture of head. Cerebrospinal fluid blood stained. Death from respiratory failure twenty-four days after onset of manifestations.*

Case 30.—31256/1911. Arthur F., aged 19, painter, single, was admitted to the London Hospital, under the care of Dr. W. J. Hadley, on June 8, and died on June 24, 1911.

Until June 1, 1911, the health of this patient had been uniformly good. On the afternoon of that day, whilst at work, he was suddenly seized with acute pain in the head "as if the head would burst" and "a feeling of stiffness all down the left side of the neck." The headache led to vomiting; the vomiting did not relieve the pain nor the feeling of stiffness. He lay down for about two hours, when the headache became less severe. He was then able to walk the quarter of a mile from his work to his home. On the next day he still suffered from severe headache over the left half of his forehead and down the left side of his neck. On June 3 he got up but did not go to work; on this day the headache, though still present, was less severe. On June 4, the pains in his head again became more severe and his appetite failed. On June 5 the headache was still severe and he experienced difficulty in opening his left eye. On the morning of June 6 he was totally unable to open his left eye. On June 7 and 8 the headache was constant and severe. On June 8 he first sought medical advice; he was seen as an out-patient by Dr. Lewis Smith and admitted to hospital.

The patient was the second of four children: his two brothers and sisters were healthy and his father and mother were alive and healthy. As a child and young man his health had been good. The only illness he remembered was an attack of scarlet fever at the age of 10 years. He denied all venereal infection and all exposure. Previous to the onset of the illness for which he was admitted he had never suffered from headache.

The patient was a well-developed healthy-looking young man of 19. He weighed 6 st. 11½ lb. He was drowsy and lethargic and only spoke when addressed. Speech was unaffected. Before June, 1911, he had never suffered from fits or other forms of seizure. He complained of a severe headache confined to the left half of his head and neck, "a dull, constant, heavy feeling, varying a little in intensity from time to time." The left side of his head was tender on pressure: the tenderness extended down over the left mastoid region and was general. Vomiting had occurred on June 1.

With the right eye the patient could see distant objects with fair ease, but with this eye he was unable to read the newspaper. Vision with the left eye was unimpaired. The arteries of the right fundus appeared normal, but the veins were engorged, whilst the disc appeared healthy; on this side there was some œdema of the lower quadrants of the fundus. On admission, the left fundus was not clearly seen, but in it no gross change was detected. Hearing, smell and taste were unaffected.

The left upper eyelid could not be raised, and of the muscles moving the left eyeball only the external rectus was not paralysed. The right eyeball was prominent but its movements were unimpaired. The right pupil was of moderate size and reacted readily to light and on accommodation. The left pupil was widely dilated and did not react either to light or to attempts at accommodation. The action of the muscles moving the jaws was normal. The right side of the face was flat and the nasolabial fold was more prominent on the left side than on the right, but in voluntary and emotional movements the face moved naturally. The movements of the palate and larynx were unimpaired. The tongue could be protruded straight and held steadily.

He walked naturally; gait was unaffected and Romberg's sign was not obtained. Whilst lying in bed he habitually held his head flexed towards the left with the chin pointing to the right. The muscles of the left side of the neck were in constant spasm. The grasps were powerful and co-ordination of the hands and legs was unimpaired. No anæsthesia or alteration in sensibility could be discovered to testing with the prick of a pin, cotton wool, the hot and cold tubes and for postures and passive movements.

The knee-jerks were readily obtained and about equal on the two sides. Ankle-clonus was not present and the arm-jerks were normal. The abdominal reflexes were readily elicited and both plantar reflexes gave a flexor response. The sphincters were under control.

The apex-beat of the heart was felt in the fifth space 2¾ in. from the mid-sternal line. The area of cardiac dulness was not increased. The heart sounds were pure. The rate of the pulse varied from 76 to 90 per minute and that of respiration from 20 to 24 per minute. No abnormal signs were found over the lungs. The temperature on admission was 100° F.; throughout the patient's stay in hospital the temperature oscillated in an irregular manner between 97° and 100° F. No abnormal signs were discovered in the abdomen; the liver and spleen could not be felt. The quantity of urine passed in twenty-four hours varied from 50 to 80 oz., the specific gravity from 1014 to 1028; neither albumen nor sugar was ever found. There was no glandular enlargement, During the whole period of observation headache was a constant feature and was not affected by the administration of drugs. The pains in the left cervical region gradually spread down the spine as far as the lumbar region, but this spread was not accompanied by any exacerbation in the headache. On June 15, the prominence of the right eye, noted on admission, became more definite and was now associated with lateral nystagmus both to the right and to the left and with coarse hippus of the pupil of the right eye; on this day, too, Kernig's sign was readily elicited on both sides.

On June 15, and again on June 18, lumbar puncture was performed; on each occasion blood-stained fluid escaped at considerable pressure, and it was observed that the longer the cerebrospinal fluid ran the more blood the specimen obtained contained.

On the morning of June 24, five hours before death, the patient's condition was closely similar to that seen on admission. He was, however, more irritable and mentally more dull than he had been on the previous four or five days. The neck muscles on both sides were rigid and the patient deeply resented all passive movements of his neck. The grasps were feeble. All the limb muscles were hypotonic but there was no absolute paralysis. The knee- and ankle-jerks could not be obtained. Both plantar reflexes gave a flexor response and the abdominal reflexes were readily elicited, and equal on the two sides. Kernig's sign was still present in both legs. On the left side the fundal veins were distended, the arteries were small and difficult to see, and the whole fundus appeared hazy; the edges of the disc were blurred and œdematous; the swelling measured more than 1 and less than 2 D.; no hæmorrhages were seen. On the right the veins were full, the arteries normal, and except for some slight blurring of the inferior and temporal quadrant the edges of the disc clear. All movements of the right eye were normal. The left third cranial nerve was completely paralysed. The left pupil was widely dilated and did not react to stimulation with intense light, the right pupil was small or of moderate size in dull light, and reacted sluggishly to intense light and doubtfully also on accommodation. The lower half of the right side of the face was paretic. The other cranial nerves were unaffected. The blood-pressure by Riva Rocci apparatus in the right brachial artery was 110 mm., in the right leg 114 mm., and in the left brachial artery 109 mm.

On June 21 the patient had had a slight convulsive seizure lasting about twenty seconds, during which time urine was voided. About 12.50 p.m., on June 24, the patient was observed to have developed stertor; no movements occurred. Five minutes later on examination he showed ptosis of both eyes of about equal severity and a bilateral divergent strabismus. The left pupil was dilated and fixed, the right of pin-point size and also fixed. Both arms and both legs were flaccid. The left knee-jerk was readily elicited, whilst the right could not be obtained, and both plantar reflexes gave a flexor response.

Three minutes later (12.58 p.m.), both arms were stiff, the right more so than the left. A knee-clonus was readily elicited on both sides, and both plantar reflexes gave an extensor response. By this time the breathing was regular and deep. At 1.1 p.m. stertor again developed and was accompanied by flexor-extensor movements of the right half of the body and of the right arm and right leg. The drooping of the upper eyelids previously noticed disappeared and the right eyeball now looked directly forwards, whilst the left still deviated outwards. Lumbar puncture was now performed (1.5 p.m.) and two test tubes full of deeply blood-stained fluid obtained. For a time this procedure seemed to give relief, but at 1.30 p.m. stertor again set in and the patient became cyanosed. For the first time since admission the palpebral fissures were now equal, but the visual axes of both eyeballs deviated widely temporalwards. The pulse was small and thready. Gradually respiration failed, the respiratory movements becoming more and more feeble and shallow in spite of the administration of oxygen. The heart was heard beating ten minutes after all apparent respiratory movements had ceased. Artificial respiration was begun, but death occurred at 2 p.m.

At the *post-mortem* examination, which took place on June 25 (P.M. 583/1911), an aneurysm measuring 1·5 cm. by 1 cm. was found at the junction of the left internal carotid and left posterior communicating arteries. This aneurysm had ruptured and a mass of blood-clot measuring 3 cm. in diameter was found over the anterior and internal subjacent portion of the left temporo-sphenoidal lobe. The meninges around the circle of Willis, more especially in the posterior fossa, were densely matted. The left third cranial nerve was stretched over and was adherent to the posterior extremity of the aneurysm. The lepto-meninges everywhere were stained brown. The lateral ventricle on both sides contained recent red clot and the surfaces of the spinal cord, more especially the posterior, were covered with dark red blood-clot. The brain weighed 1,403 grm.

The cerebral arteries appeared healthy. The heart weighed 269 grm. The valves appeared healthy; on the pericardium a few milk-spots were seen. A few fatty streaks were seen in the lower thoracic and in the abdominal aorta and at the bifurcation of the common carotid artery. The crypts of the tonsils contained muco-pus and each tonsil measured 2·3 cm. in diameter. The liver, lungs, and kidneys were congested. In the left kidney a small congenital cyst was found, and on the surface of the spleen was a small patch of perisplenitis. A small calcareo-caseous nodule was present in one of the cæcal glands.

Thus, apart from the cerebral aneurysm which had ruptured and caused death, all the organs appeared healthy. There were no signs of any cardiovascular hypertrophy, of old or recent endocarditis, and nothing to suggest any syphilitic infection.

A case of death from cerebral hæmorrhage which showed at autopsy a ruptured aneurysm at the junction of the left anterior cerebral and anterior communicating arteries and an unruptured aneurysm at the junction of the right carotid and posterior communicating arteries, with great cardiovascular hypertrophy, general arterial degeneration and atheroma of the cerebral *arteries, in which manifestations of intracranial disorder first occurred at the age of 47. Three apoplectiform attacks preceded the fatal issue. Headache, noises in the head, double optic neuritis, paralysis of the left external rectus, ptosis left. Death from respiratory failure fifty days after the onset of acute symptoms.*

Case 4.—43036/07, Matilda H., married, aged 49, was admitted to the London Hospital under the care of Dr. Francis Warner on December 14, 1907, and died on December 30.

Until 1905 her health had been good, but about two years before her death "family matters went wrong," she began to worry, became subject to attacks of severe frontal headache and suffered greatly from noises in the head and sleeplessness. In the months preceding her admission the headaches had been more frequent, more persistent and more severe. On the afternoon of November 10, 1907, fifty days before death, whilst sitting at home in her kitchen she had a "seizure"; the attack came on suddenly; "a feeling of fulness came before my eyes and was then followed by a splitting headache and severe giddiness; my sight became dim and misty, and everything looked indistinct and far away." She did not completely lose consciousness nor did she vomit. During the attack she was able to move her limbs naturally and at will, and could understand what the people around her were saying. The "seizure" lasted about five minutes; it was followed by a feeling of lassitude and inability to stir, which lasted until bed-time but did not prevent her carrying out her household duties. On the morning of the 11th she got up at the usual time and prepared breakfast, but complained of severe vertical headache; this headache persisted for three days. On November 13 she began to see double, and it was noticed that her left upper eyelid drooped; but at this time her limbs were not affected. On the 14th, four days after the seizure, she did the weekly washing for the household.

Two weeks later, on November 28, whilst at work in the washhouse, "a feeling of giddiness seized me; I sat down on a chair, then I completely lost myself and fell on to the floor; when I came to myself I was in bed; then I vomited incessantly for several days; the vomiting bore no relation to the intake of food and did not relieve the headache." She stayed in bed for twelve days. When she got up her memory for recent events, for instance as to where she had put her purse or placed things about the house, had become extremely bad. From that time until admission she complained of a constant severe headache located at the back of the eyes and over the vertex, of diplopia for distant objects, and of a dislike for bright lights. The headache was worse when the patient lay recumbent; it did not prevent sleep and was always worse shortly after waking. The ptosis of the left eye persisted unchanged.

The patient was a married woman of 49; her periods had been irregular for the six months preceding admission. Her husband, aged 51, was alive and, except that he was subject to attacks of acute gout, healthy. Her father, aged 85, was living and healthy; her mother died at the age of 65 from "asthma and bronchitis." She was the fourth of six children, her three brothers and two sisters were alive and healthy. She married at the age of 18 and had had fourteen children and two miscarriages: (i) male, living and healthy, aged 30; (ii) miscarriage at five months; (iii) female, living and healthy, aged 28; (iv) male, living and healthy, aged 26; (v) full-time child, born dead; (vi) male, living and healthy, aged 24: (vii) male, living and healthy, aged 23; (viii) male, lived three days; (ix) male, living and healthy, aged 21; (x) female, died, aged 2; (xi) female, living and healthy, aged 15; (xii) male, died, aged 1¾; (xiii) female, living and healthy, aged 12; (xiv) miscarriage of twins at 3½ months; (xv) male, living and healthy, aged 10; (xvi) female, died, aged 8 months, of "wasting." There was no history suggestive of venereal infection.

The patient was a well-covered woman of healthy appearance. Mentally she was dull and apathetic. Speech was unaffected. Memory for past events was clear and she gave her history in clear, lucid terms; she volunteered the information, however, that her memory for recent events was poor. She suffered neither from hallucinations nor delusions. She complained of a constant severe headache at the back of the eyes and over the vertex, which was most severe on waking and in the early morning. The headache did not extend into the neck or temporal regions and was not accompanied by pressure or percussion tenderness of the skull. She complained of a constant feeling of sickness which occasionally led to actual vomiting; the vomiting did not relieve the headache or the nausea, and bore no relation to the intake of food.

There was no paralysis of any limb, and to tests no alteration in sensibility could be discovered.

The knee-jerks were readily elicited and equal; ankle-clonus was not obtained. The deep reflexes of the upper extremities were normal. On each side stimulation of the sole of the foot gave rise to a brisk response; the first response was usually extensor, but then the toes would flex.

The left palpebral fissure was narrower than the right, and the left upper eyelid could not be raised voluntarily. For objects at a greater distance than 18 in. from the eyes, double vision occurred, for nearer objects single vision. The right external rectus was paralysed; all the other muscles moving the eyeballs acted normally and nystagmus was not present. The pupils were equal and reacted well both to light and to accommodation. There was some evidence of left facial paresis; the left nasolabial fold was less definite than the right, and the whole of this half of the face appeared flat; when the patient was requested to show her teeth, however, the lips moved equally on the two sides. The tongue was protruded straight and showed no tremor. The movements of the palate and larynx were normal.

The upper edge of the right optic disc was blurred and swollen; the swelling extended over the fundus on the inner side: the veins appeared normal. On the left side there was some œdema around the disc and indefinite patches of white exudate were seen on the inferior aspect of the fundus; the line of the inferior temporal artery around the disc on this side could not be traced: the other arteries were small, but on this side the veins were all slightly enlarged. The intra-ocular tension of both eyes was high. The patient complained of severe pain in the back of the eye, made worse by light. Vision with each eye was fair and the visual fields to coarse testing not grossly impaired.

Hearing was normal, but the patient complained of a noise as of "water rushing through my head." The drums appeared healthy.

The lymphatic glands were not enlarged. A heaving impulse was felt in the fifth space in the mid-clavicular line. The second cardiac sound at the aortic area was accentuated, the first sound at all areas normal and there were no bruits. The lungs were unaffected and the chest moved normally. The wall of the radial artery could be felt, but was not abnormally thick. The blood-pressure was not greatly raised. The liver and spleen could not be felt and to percussion were not enlarged. The patient was constipated. The quantity of urine passed in twenty hours varied from 12 to 44 oz. The urine contained neither albumen nor sugar and casts were not present.

The rate of the pulse varied from 68 to 76 per minute, the rate of respiration from 20 to 24 per minute. The temperature throughout was raised; on admission it was 99·8° F., before death it attained the height of 103° F.

For eight days after admission the patient appeared to improve, her headache became less severe and her mental condition brighter. On December 22, a relapse set in; she became comatose, and passed urine and fæces incontinently into the bed, whilst the rate both of the pulse and of respiration rose. Venesection on the 24th led to no improvement in symptoms or general condition. Death occurred from respiratory failure on the morning of the 30th, seventeen days after admission and seven weeks after her first seizure.

An autopsy was performed on December 31 (P.M. 1181/1907). A ruptured aneurysm, of the size of a pea, was found at the junction of the left anterior cerebral and anterior communicating arteries. Around this aneurysm old and recent hæmorrhage into the meninges had occurred. The recent hæmorrhage had caused laceration of the brain substance and caused a rupture into the right lateral ventricle. A second, unruptured aneurysm of about the same size was found at the junction of the right carotid and right posterior communicating arteries. The brain weighed 1,474 grm. The left carotid artery at its point of bifurcation was slightly dilated. The heart generally, and more especially the left ventricle, was hypertrophied. Weight of heart 453 grm. As evidence of atheromatous changes a few minute fatty plaques were observed in the intima of the basilar and right posterior cerebral arteries; a few fatty plaques in the wide coronary arteries; fatty flecks and streaks in the carotids; three large fatty, slightly calcareous buttons and numerous fatty, slightly calcareous plateaux and flecks in the aorta and a fatty patch in the aortic cup of the mitral valve. The intima of the femoral arteries was hypertrophied and the media of these vessels showed evidence of slight degeneration. The kidneys were atrophic and together weighed 270 grs. The lungs showed atrophic emphysema with hypostatic congestion.

Under the microscope the wall of the aneurysm on the anterior cerebral artery was seen to consist of fibrous and a few small shreds of elastic tissue;

no muscle was seen. The anterior cerebral artery nearer its origin showed small thickenings of the intima composed of fibrous tissue and elastic fibres; the elastic fibres appeared to have been developed from the elastic stripe. In the site of the swelling at the bifurcation of the left carotid artery the media contained no elastic fibres; the swelling itself was formed partly of muscularis and partly of fibrous tissue.

There was a general hypertrophy of the intima of the aorta; this hypertrophy was found chiefly internal to the stripe and consisted of an inner dense fibrous and an outer fibro-elastic layer. At the site of the flecks the intima was further thickened and here formed nodules. The nodular thickening occurred internal to the elastic stripe. The external portion of this thickening was occupied by atheromatous débris, which was in part calcified. The layer of longitudinal muscles and elastic was not involved in the degeneration. The structure of the buttons was essentially similar. The media everywhere showed an excess of fibrous stroma. The intima of the femoral arteries was hypertrophied. A longitudinal layer of muscles was recognized between the elastic lamina and the stripe. Internal to the elastic stripe was a zone of concentric elastic fibres. The media contained many small spindle-form areas of degeneration.

A case of aneurysm of the right intracranial carotid artery close to the junction with the right middle cerebral, which led to death from cerebral hæmorrhage after giving rise for thirteen months to chronic headache. The first leakage apparently occurred whilst the patient was at stool and was accompanied by acute headache, vomiting, pains in the abdomen and legs. Ptosis of the right upper eyelid appeared fourteen days subsequently and on the sixteenth day a second leakage was ushered in by a convulsion and was followed by acute delirium. He was admitted to hospital on the twenty-first day and at that time showed complete paralysis of the right third cranial nerve with ptosis, optic neuritis, Babinski's sign on the left side, mental disorientation, free blood in the cerebrospinal fluid and albuminuria; under observation the reflexo-motor signs varied. Death occurred from respiratory failure on the thirty-sixth day after the onset of the acute manifestations.

Case 31.—30115/08, Charles E., aged 40, grocer, single, was admitted to the London Hospital under the care of Dr. W. J. Hadley on January 16, and died on February 1, 1908.

The patient was a slight, well-covered man of 40, with dark hair and grey beard, who looked somewhat older than his years. The history of his previous health is somewhat uncertain, as at the time of admission his mental condition was curious and he had no idea of either time or space.

He stated that his health had been uniformly good until the age of 38, two years before admission; he then suffered from an attack of "appendicitis," for which he was operated upon at St. Bartholomew's Hospital, E.C. After this operation for eight months his health was fair. He then became subject to recurring attacks of occipital headache; the attacks were not severe enough to keep him from work. He denied all venereal infection and had not been addicted to alcoholic excess. For many years he had been deaf in his right ear in consequence of an attack of otitis media in childhood. He was an only child. There was no family history of vascular or nervous disease.

At 11.30 p.m. on December 27, 1907, whilst at stool, he was seized with severe abdominal pain, "shooting down my back and into my legs"; he passed a large stool and then "an electric shock seemed to run up my back into the top of my head." He managed to walk indoors but seemed "dizzy and queer." For half an hour he suffered from an intense headache and then vomited a large black vomit; the vomiting eased the pains in his head. He went to bed and immediately fell asleep. On the morning of the 28th he felt ill; a doctor was called in, who found some paresis of the patient's left leg. Between December 28 and January 16, when he was admitted, he complained of constant, severe pain in the lower part of his back, and of weakness of the legs. On January 10, ptosis of the right eye was noticed. On January 11, he suffered from a seizure which lasted for about fifty-five minutes; it came on suddenly, and in it he was "stertorous, cried out, and became stiff all over." The seizure itself was followed by shrieking and violent delirium, for which he was injected hypodermically with morphia. On the four succeeding nights he was delirious, and on the night of the 15th to 16th he had "five attacks with delirium and excitement."

On *admission*, twenty days after the onset of the illness, he made few complaints; he lay comfortably in bed and talked freely. He answered questions readily and volunteered sensible information. He had little idea of time or space; four hours after admission he stated that he had been in hospital three days: attempts to rectify this impression received the reply, "Well, I've been here three days, I know."

Speech was unaffected, and throughout his stay in hospital he did not vomit.

On the left forearm, just above the head of the radius, was a healing abrasion, brought about in his violent struggles when delirious, and over the sacrum were patches of reddened and roughened skin. The left knee was swollen, tender and slightly reddened, and the joint contained excess of fluid. The skin over the left heel was abraded. There was no anæmia and the lymphatic glands were not enlarged. On admission the temperature was 97° F., the rate of pulse 64 per minute, and the rate of respirations 30 per minute.

There was no absolute paralysis of any muscle, yet the patient was unable to turn himself in bed. All voluntary movements were carried out without precision in a clumsy, jerky fashion. The tone of the muscles of the extremities was normal and no abnormal muscular twitchings were observed. Spontaneous movements of any sort rarely occurred and under observation the left leg was never moved. He complained of no spontaneous pains and to testing with cotton wool, the prick of a pin and for the recognition of passive movement no sensory loss was discovered.

Neither knee-jerks nor ankle-jerks could be elicited. On the right side the plantar response was flexor, on the left an extensor response was obtained. Kernig's sign was not present on the left, on the right it was doubtfully obtained.

The vessels of the optic fundi were full and some œdema was seen along their course; the edges of the discs were sharp and no hæmorrhages were observed. On the right side he could neither appreciate the ticking of a watch nor the sound of a tuning-fork applied to the mastoid; both drums appeared normal. Smell and taste were unaffected.

The right upper eyelid drooped and could not be raised voluntarily, the left lids moved normally. The right external rectus muscle acted well, but the remaining muscles moving the right eyeball were completely paralysed; on the left side all ocular movements were normal. Nystagmus was not present. The right pupil was widely dilated and did not react to light or on attempted movements of this eye; the left pupil was moderate in size and reacted normally to light and on accommodation. The facial movements were normal. The tongue was protruded straight and held steadily. The movements of the palate and larynx were unaffected.

On admission the actions of the bladder and rectum were controlled and the movements of the spine were unaffected.

The apex-beat of the heart was felt in the fifth space just internal to the mid-clavicular line; the cardiac sounds were clear. There was no abnormal dulness over the lungs, and the breath sounds were normal.

A well-healed scar, 4 in. long, was seen over the right iliac fossa. The liver and spleen could not be felt, and to percussion there was no evidence of any enlargement of these organs.

The quantity of urine passed in twenty-four hours varied from 24 to 64 oz., and the specific gravity from 1021 to 1028. The urine was acid, and, on boiling, yielded a thick cloud of albumin; blood and sugar were never found. On standing, a quantity of floating mucus was deposited; this deposit consisted in the main of broken-down pus cells and squamous epithelial cells, but in it occasional hyaline casts were seen.

On January 18 lumbar puncture was performed. Cerebrospinal fluid escaped under considerable pressure. This fluid was bright red in colour and uniformly stained with blood. On microscopic examination many red cells and a few polymorphonuclear leucocytes only were found; bacteriologically the fluid was sterile.

For a few days after admission the patient's general condition improved. The headache became less severe, and his mental condition clearer; he now complained of a sensation "as if I were falling backwards." On the nights of the 17th, 18th, and 19th he was delirious; he called to imaginary persons, and received imaginary answers, but in the daytime he was more or less rational in his behaviour, though in general he lay with his eyes closed, complaining of little and speaking only when addressed. He was now able to carry out certain voluntary movements, but when left to himself performed few.

The physical signs of disease varied from day to day and from hour to hour. Sometimes on the right side a knee-jerk could be obtained, at others it was absent; at still others it was grossly exaggerated; the left knee-jerk also varied, and occasionally was absent. The plantar responses also showed variations; sometimes the right would be extensor, at others flexor; the response on the left side varied similarly, but not synchronously. The mental state gradually improved between January 19 and January 30; spontaneity returned, headache was no longer complained of, the sensation of falling backwards disappeared, and the nocturnal delirium ceased.

In the early hours of January 30 the patient was seized with severe headache; the headache was at first chiefly frontal, but soon spread backwards over the occiput and into the neck. It was accompanied by pressure tenderness of the scalp. There was, however, no rigidity of the muscles of the neck or of the back. The onset of the headache was not accompanied by any corresponding alteration in the rate of the pulse or of respiration.

At midday, on the 30th, he suddenly became worse and lost consciousness; breathing became shallow, and occasionally stertorous. The right pupil remained dilated, but the left contracted to the size of a pinhead. The rate of the pulse remained about 100 per minute.

Lumbar puncture was now performed, and cerebrospinal fluid under considerable pressure escaped in a steady stream. The fluid was fairly clear, and intimately mixed with bright red blood; two ounces were collected. The breathing became deeper and more regular, and a movement of the right hand to the head, apparently of a voluntary nature, was observed. Consciousness of his surroundings, however, did not return. Frequently repeated flexion-extension movements of the right thigh were now noted.

At 9.30 p.m. on this day he lay flaccid and unconscious. The left pupil was small, the right dilated, but not to such a great extent as heretofore. There was definite unsteadiness of the eyes on looking outwards of a nystagmoid nature. The right eye showed much greater freedom of movement than had been previously observed. Exposure of the eyes to light irritated the patient, and was followed by movements of the head and general restlessness of the body. At this time breathing was regular, and expiration was accompanied by a flapping of the cheeks and a short groan. Flexion of the right thigh was frequently seen: all the other extremities lay flaccid by the trunk. The knee-jerks could not be elicited. On the right side there was a ready extensor plantar response; on the left no plantar response could be obtained. The wrist- and elbow-jerks were absent. On the right side Kernig's sign was positive, on the left negative. No reaction to painful pressure or to the prick of a pin could be obtained from any portion of the head, trunk, or extremities. The right half of the face in both its upper and lower portions was completely paralysed, and the ptosis of the right upper lid persisted.

Eight hours later the patient's condition again became worse. Lumbar puncture was again performed, and eleven drachms of bright red fluid were obtained. After this he became steadily weaker, the breathing took on the Cheyne-Stokes' characters, and the respiratory passages filled with mucus.

Death from respiratory failure occurred twenty-four hours later at 8.30 a.m. on February 1, 1908, thirty-six days after the initial seizure.

At the autopsy performed on the afternoon of the day of death only an examination of the head was permitted. A small ruptured aneurysm, of the size of a haricot bean, was found on the right intracranial carotid artery close to the origin of the middle cerebral artery. The right temporal convolution as far as the second temporal sulcus was almost completely destroyed. Over the base of the brain and over the vertex and the region of the Sylvian fissure on the right side there was considerable subarachnoid and subpial hæmorrhage, and around the aneurysm there was a considerable quantity of older blood-clot. The lateral and fourth ventricles were distended with blood-stained fluid. Otherwise the cerebral arteries appeared quite healthy, and showed no sign of inflammation or atheromatous change.

A case in which the autopsy revealed a ruptured and partially thrombosed aneurysm of the left middle cerebral artery near the base of the brain, considerable atheroma of the cerebral arteries, severe general arterial degeneration, and slight cardiovascular hypertrophy in a woman aged 67. Eighteen years before death this patient, after complaining of left-sided local headache, had suffered a right hemiplegia with motor aphasia from which she almost entirely recovered. Seven months before death she developed ptosis and supraorbital neuralgia on the left side, the pains of which for five months were relieved by an injection of alcohol. Eighteen days before death a sudden apoplectiform seizure occurred with development of motor aphasia, œdema of the soft tissues around the left eye, complete paralysis of the left third cranial nerve and ptosis and noises in the ears; partial recovery. Seven days before death, after a seizure, paralysis of the right face, tongue, and arm, and severe hebetude appeared. Final seizure and respiratory failure on day of death.

Case 14.—40432/1913. Jane P., aged 67, married, was admitted to the London Hospital under the care of Dr. W. J. Hadley on February 11, and died on February 23, 1913. In 1891, at the age of 45, after suffering for several years with chronic left-sided headache, she had a seizure which was followed by a right-sided "hemiplegia and motor aphasia"; at this time she was "unable to talk" for fourteen days, and in bed for six weeks. The right hemiparesis completely disappeared, and except that she was still subject to recurrent episodical attacks of severe pains in the head, for the next twenty years her health was good. In February, 1912, she suffered an attack of erysipelas. In June, 1912, she was in-patient at the Croydon Hospital, complaining of trouble in opening her left eye and intense supraorbital neuralgia. For the neuralgia, alcohol was injected into the left supraorbital nerve. After the injection she was never able to raise her left upper eyelid, but the pain was relieved. About Christmas, 1912, the left supraorbital pains returned, and on December 31, 1912, the supraorbital nerve was again injected with alcohol; this injection, however, yielded no relief. On February 3, 1913, she suffered from a seizure in which consciousness was lost; on the return of consciousness she was unable to talk; she knew what she wanted to say, could write and draw, but could not utter the words. On February 4, 1913, speech returned. She then became subject to nocturnal delirium, and her ideation became inconsequent. After the seizure until admission she was kept in bed.

On admission: The tissues around the left orbit and in the left temporal region were œdematous and pitted slightly on pressure. Cerebration was slow and ideation defective. In general she lay in bed in a dazed and drowsy condition taking little interest in her surroundings. At night she was restless, noisy, and made attempts to get out of bed. To questioning she answered rationally and quickly. Speech was not affected. She was well orientated and delusions were not present. Memory was defective.

She was completely unable to open the left eye and the left upper lid drooped. The external rectus and superior oblique muscles acted normally, but the power of movement by all the muscles supplied by the left third cranial nerve was extremely feeble. The left pupil was dilated and did not react either to light or attempted convergence; the right pupil was of moderate size and reacted normally. The movements of the jaws, face, palate, tongue, and larynx were unaffected. There was considerable sensory loss over the area of skin supplied by the left supraorbital nerve, probably consequent on the injection given on December 31, 1912; hyperalgesia and loss of sensation were not discovered elsewhere in the skin supplied by the fifth cranial nerve. Hearing was unaffected, but the patient complained of a "roaring noise in both ears": the drums appeared healthy. The optic discs and ocular fundi on both sides appeared healthy. Smell and taste were unaffected.

There was no motor paresis of the limbs or trunk, and the tone of the muscles of the limbs was normal. No sensory disturbance over the body or extremities was discovered.

The knee-jerks and ankle-jerks were not exaggerated, and both plantar reflexes gave a flexor response. Neither upper nor lower abdominal reflexes could be obtained. The wrist- and elbow-jerks were normal and a jaw-jerk could be obtained.

The action of the sphincters was controlled.

The chest was emphysematous and the area of cardiac dulness small. A feeble cardiac impulse could be felt in the fifth space just inside the mid-clavicular line. The cardiac sounds were normal. The wall of the radial pulse could just be felt but the blood-pressure was not unduly high. No abnormal signs were discovered in the abdomen. The urine contained a cloud of albumin, but no sugar; its specific gravity varied from 1017 to 1025. The patient had suffered from recurrent attacks of epistaxis, but no abnormal appearances were detected in the nose, and all the sinuses on illumination appeared normal.

On the second evening after admission she was noisy, made irrelevant remarks and suffered from hallucinations and illusions. About 10.30 p.m. on this day she suddenly fell back in bed; her breathing became stertorous and dyspnœic; she lost consciousness, and for two or three minutes moved her limbs violently. After the seizure she was very irritable and restless, and yet could not move her limbs voluntarily. The puffiness around the left orbit increased. On examination complete paralysis of the left third cranial nerve, weakness of the lower half of the right face and of the right half of the tongue, and a monoplegia of the right upper extremity with increased reflexes were discovered. The right knee-jerk was more brisk than the left, but ankle-clonus was not present and both plantar reflexes gave a flexor response. On the morning of the 13th speech was impaired, but the lower extremities and left arm were under voluntary control; and the patient suffered from retention of urine with overflow. She could understand written and printed words, and attempted to answer the questions. During the whole of the 14th, respirations were of the Cheyne-Stokes type. On the 15th she talked freely but her remarks were inconsequent and nonsensical. From this time until the 21st her condition altered little; on this day she again suddenly lost consciousness and died from respiratory failure.

At the autopsy, P.M. 140/1913, performed on February 24, a ruptured and partially thrombosed aneurysm, 3 cm. in diameter, was found on the left middle cerebral artery near the base of the brain. No atheromatous changes were seen in the aneurysm itself, but the cerebral arteries generally were wide and showed severe atheromatous changes. The left third cranial nerve was small and grey in colour. The meninges in the neighbourhood of the aneurysm and in the right Sylvian fissure were deeply stained with altered blood-pigment and much matted. The myocardium everywhere was fatty. The wall of the left ventricle was 2 cm. in thickness. The base of the mitral and aortic cusps was slightly thickened. Fatty plaques and flecks were observed throughout the thick-walled aorta and calcareous plaques were seen in the aortic arch and abdominal aorta. The coronaries were wide and showed many thick-set fatty streaks and plaques, whilst the carotids were studded with fatty streaks. In the posterior parts of both lungs evidence of purulent bronchitis and hypostatic broncho-pneumonia was discovered. The kidneys were "granular;" in colour they were red, and their surfaces were irregular and showed subcapsular nodules. The cortex was narrowed, and owing to the presence of large arcuate arteries showed a tortuous pattern.

In *nine* other cases multiple seizures from partial ruptures of a cerebral aneurysm occurred.

Case 23 (p. 61) was that of a grocer, aged 41, who had been under more or less continuous observation at hospital over a period of sixteen years, and had complained of recurring attacks of headache, and been blind of an anæsthetic left eyeball for three years. The *post-mortem* examination showed that he died from the rupture of an aneurysm of the size of a currant-grape at the junction of the right anterior cerebral and anterior communicating arteries. In this patient the first bleeding occurred forty-three days, a second twenty-five days, a third seven days, and a fourth twenty-four hours before death in coma.

Case 22 (p. 64) was an example of multiple leakages and seizures due to the rupture of a small aneurysm of the left posterior cerebellar branch of the basilar artery. At the autopsy a mass of blood-clot which had pressed upon the pons, cerebellum, medulla, and the occipital lobe of the left cerebral hemisphere was discovered. The patient was a married woman, aged 42, who, when aged 25, and 8 months pregnant of her second child, had suffered a transient hemiplegia affecting the right face, tongue, arm, and leg, and who for the five months preceding death had complained of slight epileptiform (*petit mal*) attacks with visual auræ. The first evidence of definite leakage was a seizure nineteen days before death, followed by a second twelve days and a third ten days before the fatal issue. On admission to hospital the patient was delirious, and showed paresis of the left half of the face, right hemiplegia, affecting the arm and leg, and complained of tenderness in the left occipital region and stiffness of the neck; the head was held in the left cerebellar attitude, but no nystagmus was observed; in the right eye early optic neuritic changes were present.

Case 16 (p. 66) at autopsy showed a ruptured aneurysm, of the size of a pea, at the junction of the left anterior cerebral and anterior communicating arteries. The patient, a carpenter, aged 53, sustained a first seizure whilst watching a football match twenty-seven days before death. This seizure was followed by a state of delirium and a complaint of headache and cerebral vomiting; later he began to suffer from epileptiform attacks, and became clumsy in all his movements. Five days and three days respectively before death he sustained a second and a third seizure; after the third seizure he was admitted to hospital, where he died.

In Case 29 (p. 69) the rupture of an aneurysm, of the size of a pea, on the left anterior cerebellar artery caused the death of a young man, aged 22. Six days before death he sustained his first seizure, followed by a state of mental instability. Three days later a second seizure led to drowsiness, left facial paralysis, bilateral corticospinal interference, left cerebellar posture of the head, stiffness of the neck, and the appearance of free blood in the cerebrospinal fluid, and to death without any recovery of consciousness.

Case 3 was that of an upholsterer, aged 60, who died from the rupture of an aneurysm of the size of a pea on a central branch of the right middle cerebral artery. At the autopsy this patient also showed a smaller, unruptured aneurysm on a second branch of the same vessel, severe atheroma of all the cerebral arteries, severe general arterial degeneration, and great cardiovascular hypertrophy. Two years before death this patient had suffered a monoplegia of the left lower extremity, from which he soon entirely recovered; fifty hours before death a second seizure led to paresis of the left arm, abolition of the right corneal reflex, and a state of deep coma, from which he never recovered. After admission to hospital he suddenly became convulsed; after the convulsion the left-sided paresis had increased, and signs of left corticospinal interference appeared.

Case 12 was that of a woman, aged 46, who was admitted to hospital in an irritable condition after a seizure, which had come whilst she was minding her fruit stall in the street, complaining of some subjective weakness of the left upper extremity, right-sided headache, leading to nausea and vomiting, and giddiness, but showing no optic neuritis or other sign of intracranial affection. Three days after admission all her symptoms had cleared, and on the fourth day she discharged herself, to be readmitted on the fifteenth day after the initial seizure in a deeply unconscious state, which was again said to have come on suddenly, and from which she never recovered.

At the autopsy a ruptured aneurysm was found on the rightmost of three anterior cerebral arteries just beyond the origin of the anterior communicating, of the size of a pea. The convexity of the right cerebral hemisphere was covered with old hæmorrhagic clot, and in the arteries generally there was a moderate grade of degeneration and a moderate degree of cardiovascular hypertrophy. X

Case 15 was that of a married woman, aged 60, who six months before death suffered from an attack of "inflammation of the lungs," after which she never recovered her health, but became subject to recurring attacks of severe headache. The initial seizure in this case occurred whilst the patient was in bed on the seventy-fourth night before death; she woke with a sudden severe headache, leading to vomiting. This attack of headache only lasted one day, but twenty-six days later she became "dazed" after a "bad shivering attack," and in this condition was admitted to hospital. This patient showed numerous scars on the shins, apparently due to old varicose ulcers, for her serum reacted negatively to the Wassermann test. The blood-pressure in the arm measured 188 mm., and the urine contained a trace of albumen. She complained of severe frontal headache, but the fundi appeared healthy. The left face showed a slight upper motor neuronic weakness, otherwise no affection of the cranial nerves was detected. Motion, sensation, the reflexes, and the action of the sphincters were unaffected. Despite the negative Wassermann reaction the condition of this patient was thought to have a syphilitic basis, and she received an injection of 0·6 grm. neosalvarsan. After this injection the headache became more severe and more persistent, and she began to vomit several times daily. Twelve days after the injection insomnia, nocturnal delirium, "mental" incontinence of urine and fæces, and delusions, made their appearance, and the paresis of the left face became more noteworthy, whilst the quantity of albumin in the urine increased. These manifestations altered little until the day of death. On that day, whilst sitting in a chair in the ward, she suddenly fell backwards, and a few minutes later died from respiratory failure. At the autopsy all the large basal cerebral vessels were dilated, and on the right middle cerebral artery was an aneurysm of the size of a cherry-stone, which had ruptured. In the great vessels was evidence of severe arterial degeneration, and in the cardiovascular system generally some slight hypertrophy.

Two years before death a packing-case maker, aged 36 (Case 25), who for many years had been addicted to alcoholic excesses, suffered a "fainting attack, after which he was never the same man." At the time of the attack, and for three weeks afterwards, he complained of an unbearable pain in the head, which gradually became less severe, but never entirely ceased. Twelve hours and a half before death this patient was at work, when he suddenly shouted that the headache had returned, and that he felt as if the top of his head would burst; then he became giddy, seemed to lose himself, and for the next hour and a a half struggled and threw himself about. On admission he was deeply comatose and stertorous. At the autopsy an aneurysm of the size of a cherry-stone, which had ruptured, was found on the left intracranial carotid artery. In the heart and vessels few signs of disease were present, but the wall of the aneurysm itself showed atheromatous changes.

The last of this class is Case 26, that of a married woman, aged 36, who during the two preceding winters had suffered from chronic bronchitis. Three months before death she began to complain of episodical attacks of severe left frontal headache. Three days before death she was out shopping when she suddenly "came over giddy," complained of severe left temporal headache, fell in the street and was taken home and put to bed. Throughout the next three days the headache persisted, and she remained in bed at home. On the night of her death she was found deeply unconscious, having vomited, showing proptosis of the left eye and widely dilated fixed pupils, whilst the knee-jerks were abolished and both plantar responses were extensor. At the autopsy three aneurysms were found on the cerebral vessels, a ruptured aneurysm of the size of a grape-stone at the junction of the left intracranial carotid and posterior communicating arteries and two unruptured aneurysms on the right middle cerebral artery. The more proximal was the smaller and lay in the fork of a primary division of the middle cerebral, whilst the more distal, a little larger than a grape-stone, lay slightly nearer the base of the brain. Atheromatous changes were present in the peripheral portions of the unruptured aneurysms, but otherwise the cerebral arteries appeared normal, and there was only slight general arterial degeneration and no cardio-vascular hypertrophy.

IN THIRTEEN CASES MULTIPLE SEIZURES OCCURRED AT VARYING INTERVALS BEFORE DEATH.

Case	Sex	Age	First seizure	Second seizure	Third seizure	Fourth seizure	Fifth
26	♀	36	3 days	8 hours	—	—	—
29	♂	22	6 ,,	2 days	—	—	—
12	♀	46	19 ,,	16 hours	—	—	—
30	♂	19	24 ,,	3 days	2 days	—	—
23	♂	41	25 ,,	7 ,,	Few hours	—	—
16	♂	53	27 ,,	5 ,,	3 days	2 days	Few minutes.
31	♂	40	36 ,,	21 ,,	8 hours	—	—
4	♀	49	50 ,,	33 ,,	8 days	—	—
15	♀	60	74 ,,	48 ,,	Few minutes	—	—
25	♂	36	2 years	12 hours	—	—	—
3	♀	60	2 ,,	50 ,,	—	—	—
22	♀	42	17 ,,	6 months	5 days	—	—
14	♀	67	22 ,,	8 ,,	18 ,,	1 hour	—

Patients in whom abnormal conditions of the vessels are suspected are always warned against the effects of a sudden rise in blood-pressure associated with violent voluntary efforts and acute emotional experience. In this series of cases the importance of this warning is well illustrated. The manifestations of the initial rupture in Case 28, that of a young man aged 22, occurred whilst the patient was competing in a public cycle race, and in Case 16, that of a carpenter aged 53, while the patient was watching a local football match and overflowing with excitement. Case 31 sustained his first seizure whilst straining at stool; Case 5 whilst at work painting; Case 29 when at work in the stable; Case 25 when lifting a heavy packing-case; and Case 30 whilst reaching upwards to paint the top of a window. Seven examples of rupture whilst in the street occurred: Cases 1, 2, 9, 10, 12 (when minding a street-stall), Cases 24 and 26 (shopping); and in one case (Case 27) the acute manifestations developed whilst the patient was in the bathroom, and in another (Case 17) during a bout of coughing.

Although aneursym is one of the relatively common forms of "intracranial neoplasm," when the tumour is due to changes in the media of the arterial wall it grows slowly and only rarely does it attain any considerable size, hence before rupture signs of increased intracranial pressure—hebetude, headache, vomiting, and optic neuritis—are also rare. In this series of cases *in no single instance* where at autopsy the aneurysm was found to be associated with changes in the media only was there clinical evidence that the aneurysm before rupture had led to the development of *optic neuritis*. In Case 25 the patient long before the final rupture took place was blind of his left eye, but here the whole eyeball, which was anæsthetic, shrivelled. On the other hand, amongst 555 cases collected by Beadles, in 16·4 per cent. signs of "tumour only" were present, and in a further 20·7 per cent. "signs more or less suggestive of tumour" occurred before rupture. Aneurysms of embolic origin without an attendant diffuse septic meningitis have only on extremely rare occasions given rise to signs of "increased intracranial pressure," and in this series optic neuritis in association with embolic aneurysm was not noted, although in Case 9 the left disc appeared slightly swollen and the whole of the fundus on this side had a glassy appearance. Cerebral aneurysms of syphilitic origin are more frequently associated with fundal changes, but in this class of case a reactive meningitis is practically always present, and the evaluation of the relative importance of the meningitis and the aneurysmal formation as the cause of these changes is, and has been, always difficult.

In this series of cases of aneurysms due to changes in the media after rupture had occurred the condition of the optic fundi was noted in 16 cases; in 10 the discs, vessels, and fundi were pronounced healthy, and in 6 abnormalities were described.

In Case 4 (p. 30) the ruptured aneurysm was found at the junction of the left anterior cerebral and anterior communicating arteries. Here the upper edge of the right optic disc was blurred and swollen and this swelling extended over the inner side of the fundus; the arteries and veins appeared normal. On the left side there was some œdema around the disc and indefinite patches of white exudate were seen on the inferior aspect of the fundus, whilst the line of the inferior temporal artery near the disc could not be traced; the other arteries were small, but on this side the veins were all slightly enlarged.

In Case 16 (p. 66) the aneurysm was found at the junction of the left anterior cerebral and anterior communicating arteries; here the right optic disc and fundus appeared pale and the edges of the disc clearly defined, but whilst the arteries were small the veins were engorged. On the left side the disc was swollen ($1\frac{1}{2}$ D.) and its edges blurred and the veins were more engorged than on the right.

In Case 22 (p. 64) the ruptured aneurysm was found on the left posterior cerebellar branch of the basilar artery, and the patient suffered amongst other complaints from epileptiform attacks with visual auræ. In this case the left fundus was described as of healthy appearance, whilst on the right the veins were engorged, the disc pink in colour, its edges indistinct and blurred, whilst the arteries were unaffected.

In Case 27 an aneurysm on the left intracranial carotid artery, of the size of a pea, had ruptured; just before death the right optic disc was described as healthy but the left was said to be swollen.

In Case 30 (p. 26) an aneurysm measuring 1·5 cm. by 1 cm. was found at the junction of the left internal carotid and posterior communicating arteries. The right optic fundus of this patient sixteen days before death showed engorgement of the veins and some œdema of the lower quadrants, whilst on the left side no changes were detected. On the day of death the veins of the right fundus were full and the edges of the inferior and temporal quadrant of the disc blurred, whilst on the left side the whole fundus appeared hazy and the disc showed definite swelling of about $1\frac{3}{4}$ D.

In Case 31 (p. 33), a ruptured aneurysm of the right intracranial carotid artery close to the origin of the middle cerebral artery of the size of a haricot bean was found; twenty days after the initial seizure the veins of both optic fundi were large, and along their course some œdema was present, but the edges of the disc were unaffected.

Headache.—The headaches associated with the presence and rupture of intracranial aneurysms do not differ from those associated with pathological intracranial conditions owning other causes, and frequently give rise to nausea and vomiting. In general, when a patient complains of localized pain in the head, and a cerebral aneurysm is suspected on other grounds, the aneurysm will be found on the same side of the circle of Willis as the headache (Cases 12, 16, 22, 26, 27 and 30), and if the headache is worse in the frontal or orbital region it is likely that the affected vessel lies in front of the point of entry of the carotid artery into the cranial cavity (Cases 4, 15, 16, 20, 25 and 26), whilst, on the other hand, the complaint of pain localized to the occipital and upper cervical region renders the affection of a vessel in the posterior cranial fossa probable (Cases 22, 30 and 31).

In six of the 31 cases (19·3 per cent.), episodical attacks of headache were the first clinical manifestations which could be attributed to the development of a cerebral aneurysm; in all these cases at a later date the sac of the aneurysm leaked and before death signs of meningeal hæmorrhage appeared. Case 4, a woman, aged 49, for two years before death was subject to attacks of frontal headache and complained of insomnia and noises in the head. Case 27, a compositor, aged 28, for nineteen months before he came under observation suffered from severe attacks of left-sided headache, and for six months from attacks of giddiness complicating the headache. Case 31, a grocer, aged 40, complained of chronic occipital headache for thirteen months before the final ruptures; whilst Case 15, a married woman, aged 60, during the nine months before death suffered from recurring attacks of headache and during her last ten weeks showed defective cerebration. Case 26, a woman, aged 36, for three months before the final seizure complained of frontal and temporal headache limited to the left half of her head; and finally, Case 18, eight days before admission developed acute headache and nausea; she vomited several times, but not until six days later did any local signs of intracranial interference appear.

The interferences with *general cerebral functions* associated with the presence of an unruptured or of a ruptured cerebral aneurysm are not characteristic. Before rupture the blood-supply of some small portion of either hemisphere may be defective and thus produce symptoms, but this appears to be exceptional. After a partial leakage the affected vessel may become thrombosed and localized cerebral softening may thus be set up, or local destruction and ploughing up of portions of the hemispheres may be caused by the blood which has leaked primarily into the meninges. Sudden hæmorrhages are manifested by apoplectic attacks and the development of a more or less deep state of coma, and if the hæmorrhage be large, death without recovery of consciousness occurs. Smaller leakages give rise to less complete abolition of function, and thus states of hebetude, apathy, drowsiness, inattentiveness, associated with irritability, "strangeness in behaviour," "hysterical attacks," defective ideation, delirium, insomnia, nocturnal psychoses and the like may arise, and, if the surface of the cerebrum be affected and yet not destroyed, convulsions of all sorts may appear.

Hemiplegia.—The motor affections associated with ruptures of cerebral aneurysms depend upon the site of the lesion affecting the corticospinal connections, and this may be cortical, basal, or pontine. Cortical affections produce more limited manifestations than do affections at other points on the course of the pyramidal tracts. In Cases 3, 7, 15, due to an aneurysm on the middle cerebral artery, an affection of the pyramidal tract in the cortex or subcortex was noted, and in Cases 16 and 22 convulsions of an epileptiform type were associated clinically with the finding of localized damage to the cortex of the cerebral hemispheres. In Cases 3, 14 and 30 the Weber syndrome (paralysis of one third cranial nerve with crossed affection of face and limbs) was present, and in Cases 22 and 29 the pontine type of hemiplegia with facial paralysis and crossed paralysis of the limbs occurred, whilst in Cases 6, 17 and 26 pressure due to the rupture of aneurysm at the base of the brain led to bilateral interferences with the upper motor neuronic connections.

At the base of the brain the cerebral arteries bear close relation to the *cranial nerves.* One would expect, therefore, that when an aneurysm had developed upon one of the basal vessels pressure effects upon one or more cranial nerves would not infrequently occur and might prove of localizing value in diagnosis. Judged by this series of cases, however, cranial nerve affection rarely occurs until the wall of the aneurysm ruptures. In Case 14 (p. 37) alone did both the clinical history and the autopsy reveal changes of old standing in any cranial nerve; in this case the third nerve on the left side was found grey, thin and stretched over the sac of an aneurysm, 3 cm. in diameter, on the left middle cerebral artery; the aneurysm had also pressed upon the left Gasserian ganglion and caused a supra-orbital neuralgia on that side.

In *no case* was any definite evidence, clinical or pathological, of an affection of the olfactory nerves obtained.

In Case 23 (p. 61) in association with an aneurysm at the junction of the right anterior cerebral and anterior communicating arteries was a shrivelled and anæsthetic left eyeball and atrophic, grey, left optic nerve. In no case in this series was there any evidence of hemianopsia or other affection of the optic tracts, but in two cases the one recorded by Byrom Bramwell [7] and the other by Weir Mitchell [26] bitemporal hemianopsia due to the presence of a large saccular aneurysm in the infundibular region occurred.

In cases of cerebral aneurysm after rupture the third pair of cranial nerves are those most commonly involved. Ptosis, an affection of the pupils and even total paralysis of the third cranial nerve on one side, are not infrequent.

On the admission of Case 31 (p. 33) to hospital, the right upper eyelid was dropped and could not be raised, the right pupil was widely dilated and fixed, and all the muscles moving the eyeball supplied by the right third cranial nerve were completely paralysed. At the autopsy a ruptured aneurysm on the right intracranial carotid close to the origin of the right middle cerebral artery was found. Case 30 (p. 26), after a seizure, showed a similarly complete paralysis of all the muscles supplied by the left third cranial nerve; at the *post-mortem* examination this nerve was found to be stretched over and adherent to the posterior extremity of a ruptured aneurysm on the posterior communicating artery close to the bifurcation of the intracranial carotid. The patient in Case 27 was deeply comatose when admitted; in him the right pupil was small, the left widely dilated, and neither pupil reacted to the light. Here the ruptured aneurysm was found on the left intracranial carotid. In Case 26, proptosis of the left eyeball, together with fixity of the pupils, was observed; a mass of blood-clot was found at the autopsy pressing upon the left cavernous sinus; this had escaped from an aneurysm at the point of junction of the left intracranial carotid and posterior communicating arteries. Case 24 was moribund on admission; here the pupils were fixed and dilated and a rupture was found to have occurred from the right posterior communicating artery at its point of junction with the posterior cerebral. Case 23 (p. 61) after the second leakage showed severe ptosis of the right eye in addition to the atrophic left eyeball.

In Case 22 (p. 64), on admission to hospital, the left pupil was larger than the right, but both reacted normally to light; a left facial weakness and right hemiplegia affecting the arm and leg were also present in consequence of the leakage of an aneurysm on the left posterior cerebellar branch of the basilar artery. Cases 10 and 13 were deeply comatose on admission. In Case 13 neither pupil reacted to light and the size of both varied greatly from moment to moment; signs of bilateral corticospinal interference were present and at autopsy a ruptured aneurysm on the left anterior cerebral at its junction with the anterior communicating artery was found; whilst in Case 10 both pupils were immobile, and the right dilated and much larger than the left in a man, aged 55, who was admitted one hour before death; here the aneurysm which had ruptured was found on the corresponding junctional point of the anterior cerebral and anterior communicating on the right side. Case 4 (p. 29) complained of diplopia, but showed no gross abnormality of the pupillary reactions; here complete ptosis of the left upper eyelid and paralysis of the right external rectus muscles were found consequent on the rupture of an aneurysm at the point of junction of the left anterior cerebral and anterior communicating arteries and the presence of a second unruptured aneurysm at the junction of the right carotid and posterior communicating arteries.

In two cases abolition of the conjunctival and corneal reflexes was of some localizing value. In Case 3 on the right side neither of these reflexes could be elicited, and with this loss was associated a paresis of the left upper extremity owing to the rupture of an aneurysm on the right middle cerebral artery; and in Case 25 the left corneal and conjunctival reflexes were completely abolished and the right considerably diminished in consequence of the rupture of an aneurysm on the left intracranial carotid artery.

Case 14 (p. 37) during the seven months preceding death suffered from a left-sided supra-orbital neuralgia and ptosis; alcoholic injections, which were given into the nerve, relieved the acute pains for a time but the patient soon relapsed. At autopsy a firm mass of blood-clot, which had escaped from the left middle cerebral artery, was found filling the cavernous sinus and pressing upon the Gasserian ganglion. The association of major trigeminal neuralgia with the presence of a cerebral aneurysm cannot be very infrequent, for in two other instances which have come under my notice since this series was closed I have seen autopsies which revealed a similar pathological condition, in one of which before death herpes ophthalmicus developed owing to the involvement of the Gasserian ganglion in the mass of blood-clot.

Facial paralysis, due to an affection of the nerve in its intracranial course, and after its exit from the pons, usually signifies that one of the cerebellar arteries is the source of trouble. In Case 29 (p. 69), at the time of admission a stable-boy, aged 22, showed a complete, peripheral type of left facial paralysis and right hemiplegia; at autopsy a ruptured aneurysm was found on the left anterior inferior cerebellar artery. In Case 22 (p. 64) the lower half of the left face was completely paralysed and the upper half weak, whilst the right arm and leg were spastic and paretic in association with a ruptured aneurysm on the left posterior cerebellar branch of the basilar artery.

Facial paralysis of cerebral origin associated with a more or less complete hemiplegia may also occur in cases of ruptured cerebral aneurysm. Case 4, on admission, in addition to the paralysis of the left external rectus muscle and ptosis of the right eye, showed slight left facial paresis of cerebral origin; at autopsy two aneurysms were found, a ruptured one at the junction of the left anterior cerebral and anterior communicating arteries and an unruptured one at the junction of the right carotid and posterior communicating arteries. In Case 5 a right hemiplegia of arm and leg and some affection of the left extremities, together with a right facial paresis of upper motor neuron type, appeared in consequence of the rupture of an aneurysm at the point of junction of the left anterior cerebral and anterior communicating arteries.

In Case 7 an old man, aged 86, was admitted to hospital complaining of some difficulty with speech, and showing slight left facial weakness; four weeks later he died, and at the autopsy an unruptured aneurysm on the right middle cerebral artery near the base of the brain was found, and all the cerebral arteries were dilated and atheromatous. Case 15, a woman, aged 60, on admission complained of headache, giddiness, and cerebral vomiting, but the only definite sign of intracranial affection was a slight paresis of the left face, apparently of cerebral origin; at the autopsy, seven weeks later, an aneurysm which had burst was found on the right middle cerebral artery near the base of the brain. Case 30 (p. 26), at autopsy showed an unruptured aneurysm on the left posterior communicating artery; on admission to hospital this patient exhibited a complete paralysis of the left third cranial nerve and grave paresis of the right half of the face.

Deafness, as the consequence of an unruptured cerebral aneurysm, is not common. Case 20, a man aged 47, died in hospital of acute diffuse myelitis. Some months before death this patient suffered an "influenzal attack," which left him subject to severe frontal headache, and deaf of his right ear. At the autopsy an unruptured aneurysm of the size of a pea was found at the point of bifurcation of the right intracranial carotid artery.

Case 31, on admission, in addition to a complete paralysis of the right third cranial nerve, showed an almost total nerve-deafness of the right ear; the aneurysm which had ruptured was found on the right intracranial carotid close to the origin of the right middle cerebral artery.

Case 4 was the only example in this series of a patient who complained of "noises in the head and ears"; here a ruptured aneurysm was found on the left anterior cerebral artery at its junction with the anterior communicating and a smaller unruptured aneurysm at the junction of the right carotid and posterior communicating arteries. In the literature of these cases buzzing noises in the head, hissing noises in the ears, waterfall noises, and even murmurs audible to the doctor, have been described.

In no single case in this series was any affection of the ninth, tenth, eleventh, or twelfth nerves below their nuclear origin discovered; and in no case did polyuria or glycosuria occur.

Pyrexia.—After a seizure consequent upon the rupture of the sac of a cerebral aneurysm of non-embolic origin, some degree of pyrexial disturbance is almost the rule, but in general the temperature reached is not high.

In Case 22 (p. 64) a rupture of an aneurysm on the posterior cerebellar branch of the basilar artery led before death to the development of a temperature of 105° F.; and in Case 18 the rupture of a similar aneurysm on the left anterior communicating artery to a temperature of 106° F.; but in no other case in this series did the thermometer record more than 103° F. In *five* cases (5, 6, 12, 13, and 29) the highest record was between 102° and 103° F.; in *two* cases (16 and 23) the highest point reached was 101° F., and in *six* cases (4, 14, 25, 27, 30, and 31) no temperature higher than 100° F. was observed, and in *ten* cases (2, 3, 9, 10, 15, 17, 21, 24, 26, 28) no record showing a higher figure than 99° F.

Stiffness of the neck, after the rupture of a basal cerebral vessel, is a not uncommon manifestation and source of complaint. The appearance of this symptom implies that blood has leaked into the posterior cranial fossa, and that clot has surrounded and compressed the lower medullary and upper cervical nerves. The presence of this symptom, therefore, renders probable the finding of the aneurysmal sac either on the basilar artery (or one of its branches) or on the posterior communicating artery. In general, however, the stiffness is both less troublesome to the patient and less persistent than it is in cases of basal meningitis. In Case 22 (p. 64) all the muscles of the back of the neck became painful and stiff, and the patient complained of severe occipital headache after the rupture of an aneurysm on the left posterior cerebellar branch of the basilar artery. In Case 29 (p. 69) a ruptured aneurysm on the left anterior cerebellar artery caused a similar complaint of stiffness of the neck, and made the patient hold his head flexed upon the left shoulder in the pose characteristic of a cerebellar lesion of the left side. In Case 30 (p. 26) the first symptom of which the patient complained was a left-sided cervical stiffness, accompanied by intense pain and tenderness of the left occipital region. During the next twenty-four days these symptoms persisted. After death an aneurysm, which had leaked and allowed blood to escape into the basal cisterns, was found on the left posterior communicating artery near the bifurcation of the carotid.

At the present time, in cases of meningitis, the only certain signs of an inflammation of the membranes covering the cerebrospinal axis is the finding of organisms or of cellular exudate and products in the cerebrospinal fluid; just so, in cases of hæmorrhage into the brain and spinal cord, the only certain proof that some vessel has ruptured is the finding in the fluid removed by lumbar puncture of blood-cells and pigment more or less uniformly intermingled with the cerebrospinal fluid. Moreover, it is found that if the blood has escaped from a vessel in the cranial cavity, the longer the fluid after lumbar puncture is allowed to run, the more blood-stained the specimen becomes; whereas, if at the time of the insertion of the needle damage is done locally to a vessel in the lumbar sac, the first few cubic centimetres of fluid collected are found to contain the greatest amount of blood pigment and the greatest number of cells. If the hæmorrhage is of recent origin, the cerebrospinal fluid shows bright red staining and little altered red blood-cells; later the colour becomes paler and yellower, and the red cells more broken up.

In Case 5, on the day of the seizure, the cerebrospinal fluid obtained by lumbar puncture showed much bright red staining and many little altered red blood-cells; at autopsy this blood was found to have escaped from a burst aneurysm at the junction of the left anterior cerebral and anterior communicating arteries. On the day of death the cerebrospinal fluid of Case 29 (p. 69) was found to be under considerable pressure and to contain a few red cells, an excess of polynuclear cells, but no organisms, and to be stained slightly with recent, little altered, blood pigment. The blood in this case had escaped from an aneurysm on the left anterior inferior cerebellar artery. In Case 30 (p. 26) the cerebrospinal fluid was examined on three occasions—fifteen days, eighteen days, and twenty-four days after the first seizure; on each occasion the fluid was under considerable pressure, and contained much blood pigment and many red blood cells; and on each occasion it was noted that the later specimens obtained after lumbar puncture contained more pigment than did those first collected.

As a point in differential diagnosis, it is noteworthy that in cases of cerebral aneurysm the vessel whose wall gives way lies in the meninges outside the parenchyma of the central nervous axis, whereas in cases of direct rupture or of rupture into a cerebral or cerebellar neoplasm the vessel gives way in the substance of the parenchyma, with the consequence that in aneurysm staining of the cerebrospinal fluid occurs at an earlier moment, and before the leakage is so profound. Moreover, as Wichern has noted, the finding of blood-stained cerebrospinal fluid is more likely to be of aid in early diagnosis if the aneurysm occurs on one of the vessels of the posterior cranial fossa.

CHAPTER IV.—THE CLINICAL MANIFESTATIONS ASSOCIATED WITH THE DEVELOPMENT OF EMBOLIC INTRACRANIAL ANEURYSMS.

Clinically, cases of intracranial aneurysm of embolic origin fall into a different nosological group from those intracranial aneurysms of congenital and degenerative origin. In the embolic class clinical evidence of a state of septicæmia and general infection is usually present when the patient first comes under observation, and in the great majority of cases, as Church [8] first pointed out in 1870, evidence of cardiac disease, of the ulcerative or progressive type, has preceded the onset of the cerebral manifestations. Between 1907 and 1913 at the London Hospital thirteen examples of embolic cerebral aneurysms occurred, and in these a history of several attacks of acute rheumatism followed by the development of a condition of progressive endocarditis was obtained in seven cases (Cases II, III, IV, VII, VIII, X and XII) and at autopsy pathological evidence of infected endocarditis was also found in Cases I, V and IX. Case VI was one of pyæmia subsequent to a retropharyngeal abscess, Case XI was one of pyæmia where no primary focus could be determined even at the *post-mortem* examination, and Case XIII one of puerperal septicæmia with general pyæmia.

In two cases only (Case I, p. 70, and Case IX, p. 71) did the patients first come under observation at hospital complaining of nervous manifestations and in both before death signs of valvular disease of the heart were discovered, and in both in addition a history was obtained of indefinite aches and pains preceding by some days the onset of the hemiplegic seizure.

Blood cultures were taken in Case III on three occasions with negative results; the blood in Case IV yielded a Gram-negative diplococcus, in Case V a short-chained streptococcus, in Cases VII and VIII a streptococcus and in Case XI a staphylococcus. In the other seven cases no blood cultures were made.

In Cases VI and XIII the patients were extremely ill, and although under observation as in-patients at hospital, at the time of death no local cerebral lesions were suspected. At the autopsy in Case VI a thick-walled unruptured cerebral aneurysm was discovered on the left anterior cerebral artery, and in Case XIII two ruptured aneurysms on small cortical vessels were found. Case XI before death fell into a severe typhoidal state, and although meningitis was suspected and thought to be consequent on a pyæmic abscess, no localizing nervous signs were discovered; in this patient the *post-mortem* examination revealed an aneurysmal dilatation and blockage at the bifurcation of the basilar artery.

Ruptures of embolic cerebral aneurysms leading to apoplectic attacks in patients who did not realize that they were ill occurred in Case I, a small girl of 7, whilst playing in the street; in Case VII, a man of 31, in the street when returning from work; and in Case IX, a girl of 15, whilst helping with house work.

Case I (p. 70) was admitted unconscious to the surgical side and gave the signs of a left hemiplegia: she was at first thought to be suffering from a "fractured base," but on the discovery of mitral disease a correct diagnosis was made before her death, which occurred six hours after the apoplectic insult.

Case VII was found lying in the street unable to speak and paralysed down the left side of the body; he was brought up to the hospital by the police, where eighteen days later he died. The first attack of acute rheumatism in this patient occurred sixteen years before death, and during the two and a half years before the cerebral infarction, breathlessness, headache, and joint pains had been extremely troublesome. At the autopsy a ruptured septic cerebral aneurysm with thick walls, of the size of a cherry, beneath the inferior extremity of the right ascending parietal convolution was discovered.

The clinical history of Case IX (p. 71) is most illuminating. She was admitted to hospital with a right hemiplegia and total aphasia, but showing no signs of cardiac disease. Lumbar puncture yielded sterile blood-stained cerebrospinal fluid, suggesting that some vessel supplying the central nervous system had leaked. As an in-patient the hemiplegic weakness largely disappeared and the power of speech returned, but twenty-two days after the seizure signs of mitral disease were recognized and she began to complain of abdominal pain. During the next week she vomited frequently, wasted and became very anæmic. No definite abdominal signs were ever discovered and she died thirty-two days after the initial hemiplegic seizure apparently from exhaustion. At autopsy a ruptured aneurysm of the cœliac axis artery ruptured into the lesser peritoneal sac, a thrombosed aneurysmal dilatation of the left renal artery, a thrombosed aneurysmal dilatation at the bifurcation of the left intracranial carotid artery and progressive endocarditis without cardiac hypertrophy were discovered.

In Case X, the patient was at home in bed when he suffered his first seizure, and in Cases II, III, IV, V, VIII and XII the first nervous manifestations occurred whilst the patients were under observation and treatment in hospital for progressive endocarditis. Case II, a man, aged 41, with an extremely large heart and ever-changing signs of mitral and aortic disease, had been in hospital fourteen days, when he woke at 7 a.m. one morning feeling weak and faint ; at 7.30 a.m. he suddenly fell back in bed complaining of weakness of the right arm, became comatose and died thirty hours later from a ruptured septic aneurysm on the right anterior cerebral artery. Case III, a boy, aged 16, had been in hospital nine and a half weeks suffering from increasing myocardial weakness and pyrexia, when he was suddenly seized with acute pain in the left side of his head. He immediately lost the power of speech and became hemiplegic on the right side. Consciousness was soon lost, and one and a half hours after the seizure with signs of respiratory failure he died. At the autopsy a septic aneurysm of the size of three peas, which had ruptured, was found on a large temporal branch of the left middle cerebral artery. Case IV, a young man, aged 22, had been in-patient three and a half months and had suffered from many attacks of pain due to the infarction of the organs, when fifty hours before death he suffered a slight generalized convulsion, became comatose and died. At autopsy an unruptured septic aneurysm at the anterior superior angle of the island of Reil on a branch of the right middle cerebral artery was found. Case V came into hospital complaining of cardiac disease and pain in the left upper thigh ; eight days before death he was suddenly seized with intense pain above the left orbit. This pain lasted acutely for a few hours, and five days later, accompanied by transient blindness of left eye which lasted a few hours only, returned. Fifty-six hours before death he became distressed, vomited, lost consciousness and developed signs of a left-sided alternate hemiplegia, paralysis of the left third cranial nerve and paralysis of the right face, tongue, arm and leg. Two septic cerebral aneurysms were found at the autopsy, a ruptured one on the left anterior perforating artery and an unruptured one on a branch to the parietal lobe of the left middle cerebral.

Case VIII, a girl, aged 16, after a three months' stay in hospital, suddenly became comatose and died three days later from a ruptured aneurysm on the right middle cerebral artery, without ever recovering consciousness. Case XII, a man, aged 36, had been fourteen days in hospital, when for some hours in the evening he became restless and excited, and after the attack was found to have a dilated left pupil which reacted sluggishly to light; nine days later on waking about 6 a.m. he became loquacious, restless, and complained of a roaring noise in his head; two or three minutes later he lost consciousness and when seen at 8 a.m. was comatose and showed evidence of right-sided cortico-spinal interference. Thirty hours after this seizure he died, and at autopsy a large ruptured aneurysm, 2·5 cm. in diameter, on the left middle cerebral artery at the end of the horizontal limb of the Sylvian fissure, was discovered.

After the rupture of the aneurysm on the right middle cerebral artery in Case I a spastic left hemiparesis, convulsive movements of the right extremities and conjugate deviation of the eyes alternately to right and left were noted. In Case II an apoplectic seizure due to the rupture of an aneurysm on the right anterior cerebral artery led to a complete paralysis of the right arm and weakness of the right leg, with signs of bilateral pyramidal interference, and a fixed dilated pupil on the right side. Case III had suffered since early childhood from weakness of the left external rectus muscle and middle ear deafness on the left side. After the rupture of the aneurysm on a branch of the left middle cerebral artery speech was lost completely and the right face, arm, leg, and right upper eyelid became paralysed. In Case V, after the seizure due to the rupture of a septic aneurysm on the left anterior perforating artery, the left upper eyelid was completely paralysed, the power of speech was lost and paralysis of the right face, arm and leg was noted. Case VII was first seen twelve days after the seizure; at this time he could utter some words distinctly but could not propositionize and got angry when he could not answer simple questions of which, apparently, he knew the answer. He could copy simple written characters and diagrams and made attempts to draw common objects; he could not copy sentences or printed words. He complained of right parieto-occipital headache. The left arm and left leg were paralysed and the movements of the left face, left half of the palate and tongue were impaired. Signs of cortico-spinal interference to the left half of the body were present and sensibility on this half was altered. Gradually these signs improved and the patient became able to repeat any simple combination of words. Four days before death he developed a nocturnal psychosis with excitement and next morning speech was again limited to a few words, which, though constantly uttered and misused, could not be repeated on demand. From this time on, his general condition got worse and for thirty-six hours before death he was comatose. The rupture had occurred near the temporal pole from a branch of the right middle cerebral artery. After a seizure Case IX became hemiplegic on the right side, showed paresis of the right face, became unable to put out her tongue, and developed proptosis of the left eyeball and optic neuritis on this side.

The first seizure in Case X occurred nine days before the patient came under observation and was followed by weakness of the left arm and some difficulty with speech. When admitted to hospital two days after a second seizure he was unconscious, and exhibited a hypertonic left arm, a spastic left leg, a flaccid right arm and a hypotonic right leg, paresis of the left face and evidence of bilateral pyramidal interference; the right pupil was dilated, the left of medium size: neither reacted to light. He died without ever recovering consciousness seven hours later. At the autopsy a partially thrombosed and ruptured septic aneurysm of the size of a cherry-stone was found on the right middle cerebral artery 5 cm. from its origin.

In the paper quoted above Sir John Rose Bradford [4] remarks: "My point is that a patient may die from cerebral aneurysm which may have resulted from embolism, but that at the time of death you may not be able to find any gross evidence of its embolic 'origin.'" If Case IX (p. 71) had died shortly after her admission to hospital, certainly progressive endocarditis would not have been diagnosed, for between January 15 and February 6, 1911, repeated examinations of the chest by a series of competent observers failed to reveal any abnormal cardiac signs; no murmurs were present, she was not short of breath and the extent of the projection of the heart against the chest wall was certainly not increased. After the latter date, however, unequivocal signs of mitral disease appeared. At autopsy there was endocarditis of both the mitral and aortic valves, but the size of the heart was not increased.

In the cases of cerebral aneurysms due to infected emboli, apart from the characters of the hemiplegias as signs of localizing value we had in Case IX unilateral (left-sided) proptosis and optic neuritis; the unruptured aneurysm was found at the bifurcation of the left intracranial carotid artery pressing upon the left cavernous sinus. Case V was a good example of the alternate hemiplegia known as the Weber syndrome, left-sided ptosis and paralysis of the right face, tongue, arm and leg. Case VII showed a characteristic interference with internal speech and apraxia of the right upper extremity; a ruptured aneurysm was found near the temporal pole on the main branch of the right middle cerebral artery.

Many authors have laid stress upon the appearance of subjective noises in the head; in Case XII alone of this series is any mention made of this manifestation. This patient, before he lost consciousness, complained of an intense "roaring in the head"; here a large aneurysm 2·5 cm. in diameter was found at the end of the horizontal limb of the left Sylvian fissure.

CHAPTER V.—SUMMARY OF CONCLUSIONS.

(1) The clinical and pathological findings in 44 new cases of cerebral aneurysm are described.

(2) From the point of view both of the clinical history and the *post-mortem* appearances, the cases showing at necropsy aneurysmal formations on the basal cerebral arteries fall naturally into two groups, the non-inflammatory and the inflammatory.

(3) In a series of 5,432 consecutive examinations of the head cerebral aneurysms due to non-inflammatory changes in the middle wall of an artery occurred in 0·57 per cent., and aneurysms due to inflammatory changes in the cerebral arteries associated with infective embolism in 0·24 per cent.

(4) In the non-inflammatory group degenerative changes in the walls of the basal cerebral arteries were often found associated with high blood-pressure, cardiac hypertrophy and evidence of general arterial disease, but in many of the cases there was no evidence of excessive blood-pressure either at autopsy or from clinical observations, and the general arterial degeneration and the degeneration of the cerebral arteries was no greater than in the average subject of the same age.

(5) In the non-inflammatory group a congenital weakness of the arterial wall at junctional points is an important factor in their formation.

(6) The proportion of aneurysms associated with infective embolism in the cerebral arteries to those in all the other arteries of the body was as 15 to 9.

(7) In this series of cases no example of a cerebral aneurysm due to, or associated with, an infection by the *Spirochæta pallida* occurred, but 96·2 per cent. of the aneurysms of the aorta, 100 per cent. of the aneurysms of the other elastic arteries, and only 6·25 per cent. of the

aneurysms of the muscular and small elastic arteries, to which class the arteries of the circle of Willis and the cerebral arteries generally belong, were consequent on the local weakening of the arterial wall due to the inflammatory reaction set up by the activity of the *Spirochæta pallida.*

(8) Rupture of a cerebral aneurysm led to death in 0·44 per cent. of all cases of examination of the body, whilst direct rupture of an intracranial artery during the same period was the cause of death in 0·91 per cent.

(9) The largest aneurysm measured 30 mm. in diameter, but the great majority were much smaller.

(10) In 15 out of 31 cases (48·4 per cent.) of non-inflammatory cerebral aneurysms, whose ages varied from 53 to 19 years and averaged 38, no cardiovascular hypertrophy was present.

(11) The age-incidence of cerebral aneurysms of embolic origin is determined by that of progressive infective endocarditis and other chronic septicæmic and pyæmic conditions and is lower than in the non-inflammatory group.

(12) In 25 out of 31 cases (80·7 per cent.) of non-embolic aneurysm clinical manifestations attributable to cerebral hæmorrhage occurred; in 5 out of 31 (16·1 per cent.), no clinical manifestations of intracranial disease were recorded, and at autopsy an unsuspected and unruptured aneurysm was found; and in 1 case an unruptured aneurysm at the junction of the right carotid and middle cerebral arteries was found at the necropsy on a patient who for some time had suffered from frontal headache and had been deaf of his right ear.

(13) Amongst the cases which at autopsy showed ruptured non-embolic aneurysms two patients died before admission to hospital, and ten were admitted after a first apoplectic seizure and died without ever recovering consciousness.

(14) Multiple leakages of blood due to partial ruptures of the aneurysmal sac occurred in 13 out of 31 cases (41·9 per cent.). In these patients a history of multiple seizures of an apoplectic type occurred.

The clinical manifestations in this type of case form a definite nosological group rendering their diagnosis not difficult.

(15) The first rupture of the sac of a cerebral aneurysm was often brought about by a violent muscular effort or acute emotion.

(16) Before the aneurysmal sac ruptured signs of intracranial pressure were rare, but after this had taken place frequent; 10 cases out of 31 before death showed changes in the optic fundi.

(17) The headaches associated with non-embolic cerebral aneurysms have some localizing value.

(18) The interferences with intellectual and reflexomotor functions which occur in association with cerebral aneurysms, both before and after rupture, depend upon the site of the vascular deficiencies and the course which the hæmorrhage takes after rupture has occurred.

(19) The cranial nerves are frequently involved in blood-clot after rupture. Optic atrophy, bitemporal hemianopsia and lesions of the optic tract have been described, but the third pair of cranial nerves were those most commonly involved in this series of cases. Supraorbital neuralgia may be caused by pressure upon the Gasserian ganglion. In cases of aneurysms of the posterior fossa, and more especially of the cerebellar arteries, an involvement of the facial nerve after its exit from the pons was common. A subjective complaint of noises in the head occurred only in one case, and in this series no local affection in the posterior fossa of the ninth, tenth, eleventh, or twelth pair of nerves was noted.

(20) After rupture of aneurysms of the posterior fossa, a complaint of stiffness of the neck is a sign valuable in diagnosis.

(21) The finding of blood cells and blood pigment in the cerebrospinal fluid is the only clinical proof of cerebral hæmorrhage; in ruptured aneurysms staining of the cerebrospinal fluid occurs early.

(22) Of 13 cases of aneurysms on the cerebral arteries due to infective embolism, 10 were associated with ulcerative, progressive endocarditis, and in 5 cases a positive blood culture was obtained, but in 2 cases the onset of nervous manifestations first caused the patients to seek admission to hospital.

(23) Rupture of the sac of an embolic cerebral aneurysm leading to cerebral hæmorrhage and the death of the patient occurred in 9 out of 13 cases.

(24) The localizing signs in cases of embolic aneurysms are usually scanty.

LITERATURE.

[1] Bartholow. *Amer. Journ. Med. Sci.*, 1872, vol. lxiv, p. 373.
[2] Beadles. *Brain*, 1907, vol. xxx, p. 285.
[3] Bradford. *Brit. Med. Journ.*, 1894, vol. ii, p. 869; *Trans. Clin. Soc.*, 1895, vol. xxxviii, p. 1.
[4] *Idem. Lancet*, 1908, vol. ii, p. 703.
[5] Brinton. *Trans. Path. Soc.*, 1852, vol. iii, p. 49.
[6] Bruns. "Die Geschwülste des Nervensystems," 1908, ii Aufl., S. 59.
[7] Byrom Bramwell. "Clinical Studies," July 2, 1906, p. 289; *Rev. of Neurol. and Psychiat.*, 1906, vol. iv, p. 507.

[8] Church. *St. Bartholomew's Hosp. Reports*, 1870, vol. vi, p. 99.

[9] Eppinger. *Arch. f. klin. Chirur.*, 1887, vol. xxxv, Supplem. S. 1.

[10] Gowers. "Diseases of the Nervous System," vol. ii, second edition, 1893, p. 529.

[11] Gull. *Guy's Hosp. Reports*, 1859, vol. v, p. 281; New Sydenham Society's Publications, 1894, vol. cxlvii, p. 86.

[12] Hale White. *Trans. Clin. Soc.*, 1895, vol. xxxviii, p. 1.

[13] Hofmann. *Zeitschr. f. Morphol. und Anthropol.*, 1900, vol. ii, S. 247.

[14] Langbein. *Münch. med. Wochenschr.*, 1913, vol. lx, S. 22.

[15] Lebert. *Berlin. klin. Wochenschr.*, 1866, vol. iii, S. 60, 209, 229, 249, 282, 336, 345, 387, 402.

[16] Newton Pitt. *Brit. Med. Journ.*, 1890, vol. i, p. 827.

[17] Osler. "Principles and Practice of Medicine," eighth edition, 1912, p. 982.

[18] Osler and Macrae's "System of Medicine," 1908, vol. iv, p. 459.

[19] Parnisetti. Quoted by Rothmann.

[20] Peacock. *St. Thomas's Hosp. Reports*, 1876, new series, vol. vii, pp. 119 and 317.

[21] Ponfick. *Virchow's Arch. f. Path., Anat. und Physiol.*, 1873, vol. lviii, S. 528.

[22] Rothmann. *Arch. f. Psychiat.*, 1904, vol. xxxviii, S. 278.

[23] Simmonds. *Deutsch. med. Wochenschr.*, 1901, vol. xxvii, S. 353.

[24] Turnbull. *Quart. Journ. Med.*, 1915, vol. viii, p. 201.

[25] von Hofmann. *Wien. klin. Wochenschr.*, 1894, vol. vii, S. 823, 848, 867, 886.

[26] Weir Mitchell. *Journ. Nerv. and Ment. Dis.*, 1889, vol. xvi, p. 44.

[27] Wichern. *Münch. med. Wochenschr.*, 1911, vol. lviii, Nr. 51, S. 2224.

[28] *Idem.* *Deutsch. Zeitschr. f. Nervenheilk.*, 1912, vol. xliv, S. 220.

[29] Windle. *Journ. Anat. and Physiol.*, 1888, vol. xxii, p. 289.

[30] Wyrubow. Quoted by Rothmann.

APPENDIX.

(A) Non-Embolic Aneurysms.

A case in which death occurred from the rupture of an aneurysm at the junction of the right anterior cerebral and anterior communicating arteries in a man aged 41, who had been under more or less continuous observation at hospital over a period of sixteen years, and had complained of recurring attacks of headache and been blind of an anæsthetic left eyeball for three years. The first seizure due to the rupture of the sac occurred forty-three days, a second twenty-five days, a third seven days, and a fourth twenty-six hours before death in coma.

Case 23 (32377/1903, 30306/1906, 30181/1909).—Edward C., aged 41, grocer, single, was admitted to the London Hospital under the care of Dr. Cecil Wall on January 21, 1909, and died on January 25. He had been under observation and in-patient at various times during the preceding twelve and a half years. In 1893, at the age of 25, he attended as an out-patient and was treated for "colic and left wrist-drop" with aperients, electricity, and liniments. At this time the case was diagnosed as one of "(?) plumbism"; under treatment his symptoms disappeared. In 1896 he was readmitted suffering from chronic diarrhœa; after seven weeks' treatment he was discharged relieved. From this time until his death he was subject to episodical attacks of diarrhœa and melæna. In 1903 enteroptosis was diagnosed, and he was treated with massage. In the full histories taken on these admissions no mention is made of any headache or abnormality of the left eye. Between 1902 and 1903 he suffered two attacks of hæmatemesis, and during the years 1896 to 1903 his weight varied greatly, between 11 st. 7 lb. and 8 st. 4 lb.; he stated that when his weight was low he was subject to attacks of diarrhœa and left-sided abdominal pain.

In January, 1906, he began to complain of headache, sleeplessness, and a feeling of confusion immediately after waking, in addition to his old symptoms of flatulence, attacks of diarrhœa alternating with periods of obstinate constipation and left-sided abdominal pain. A note made at this time states that he was blind of the left eye, following iridectomy for staphyloma, but does not give the date of the operation, and further states that the cornea of the shrivelled eyeball showed scars of old corneal ulcers. At this time no abnormal nervous signs were discovered; the heart was not enlarged and the cardiac sounds were clear. An explanation of his symptomatology was found in the profound enteroptosis and the finding of a right movable kidney and occasional albuminuria. The stomach was greatly dilated and prolapsed; when the patient stood erect the small intestines fell forwards and the hypogastrium bulged; the transverse colon could be moved vertically over a distance of 3 or 4 in. Under massage and hospital treatment his weight in sixteen days rose from 8 st. 4 lb. to 9 st. 1 lb. On discharge he was given an abdominal belt, and three months later reported himself as "free from all the old symptoms except the headache."

On December 12, 1908, forty-three days before death, he was seized with a "bad influenza"—dreadful headaches day and night, pains in the back of the head and neck, constipation, and vomiting. He remained in bed four days. During these days he retched frequently, his motions were loose and he complained of severe frontal and vertical headache. For the next two weeks he was constantly ailing; the headache never ceased, but he was able to attend to his shop. On December 31, 1908, twenty-five days before death, in the morning about 4.30 a.m., the headache became "worse than ever," he felt "really bad in himself," got out of bed and "started cursing everybody, using very foul language." After this attack until death he did no more work and complained of headache "always present but varying in intensity from day to day and hour to hour." On January 17, 1909, he suffered another exacerbation in symptoms. The pain in the head on this day seemed to begin in the back of the head just beneath the hairy region and shoot forward to between the eyes and "almost push my eyes forward." On the 19th he vomited several times quite apart from the intake of food, which he usually refused, and became extremely constipated. On the night of January 20 to 21 he became very excited, cried out for a knife with which to murder his wife, could not be kept in bed, and beat his children. He was given injections of morphia, and next morning admitted to hospital.

On admission.—The patient was a thin, anæmic man weighing 7 st. 8 lb He was extremely irritable, resenting all interferences and exposure to light.

At times he was noisy and called for objects with which to commit murder on his relations. In a general way he was intelligent, talked freely and answered questions lucidly. He could not give any connected account of his actions and mode of life during the preceding weeks, but readily answered questions relating to his shop and previous admissions to hospital. He complained of much severe left-sided frontal headache and said that the pain was increased by pressure between the eyes. After admission he vomited frequently; the vomits were small, bile-stained and consisted almost entirely of altered and clotted milk; the vomitings bore no relation to the intake of food.

Speech was unaffected and attention feeble, his remarks flitting rapidly from thought to thought. There was no paralysis or weakness of any extremity. The grasps were equal and powerful. He complained of no spontaneous pains in the limbs and to testing no loss of tactile, painful or thermal sensibility was discovered. The answers to tests for the recognition of posture and passive movement were poor and defective compared with those obtained in other modes of examination. No trophic or vasomotor changes were noted.

The knee-jerks were glib and equal on the two sides. Ankle-clonus was not obtained. No plantar responses were obtained. The arm-jerks were natural.

The left eyeball was small and atrophic, and with this eye the patient could not appreciate light. The left fundus could not be seen; the right appeared healthy, and the edges of this disc were clearly seen and sharply defined.

There was definite ptosis of the right eye; this eyeball moved badly but no local weakness of any single muscle could be determined. Nystagmus was not present. The right pupil was small, regular in outline, and reacted normally to light and convergence. The movements of the face, jaw, tongue, palate and larynx were unaffected.

The patient suffered from retention of urine, and catheterization was necessary; the bowels were only opened after the administration of enemata.

The tongue was dry and the pharynx filled with dried mucus. The chest was long, flat, phthinoid, the intercostal spaces wide and oblique, the sub-costal angle narrow, and the costal margins everted; a Harrison's sulcus was present and the sternum was depressed. The neck was thin and the trapezii long and sloping. The apex-beat of the heart was felt in the fifth interspace in the mid-clavicular line. No murmurs were heard. There was no abnormal dulness over the lungs and the breath sounds were natural.

The abdominal wall was thin and the distance between umbilicus and pubes long; the outlines of the stomach which filled the epigastric and upper umbilical region could be seen, and splashing was easily elicited. The liver and spleen were not enlarged. The right kidney was easily felt and could be seized between the fingers. The urine contained a trace of albumin, but no sugar.

On the morning of January 24 the patient suddenly cried out, "Lift me up, it's coming again." A nurse rushed up to him and lifted him up; then he began to jerk his arms, head and legs. He did not bite his tongue. His colour changed and the pulse became feeble and rapid. After about half a minute the movements became more violent, then general rigidity followed by a state of deep unconsciousness and snoring breathing set in. He remained stiff for some minutes and gradually fell into a state of heavy sleep. After the seizure he was found to have passed urine and fæces under him. During the next twenty hours he occasionally raised his right arm and waved his right hand or picked with it at the bedclothes. No absolute paralysis appeared, but the left arm became more flaccid than the right. On January 25, twenty-six hours after this seizure, he died of respiratory failure.

At the *post-mortem* examination made on January 26 (P.M. 87/1909), a ruptured aneurysm of the size of a currant-grape was found at the junction of the right anterior cerebral and anterior communicating arteries, from which blood had escaped, covered the frontal lobes, filled the superior longitudinal fissure and the basal cisterns, and had spread over the whole of the cerebellum. The frontal lobes were stained brown. The substance of the hemisphere around the aneurysm was softened and had allowed blood to escape into the lateral, and thence into the third and fourth ventricles. The brain weighed 1,545 grm. The left optic nerve and left eyeball were atrophic. In the aneurysm itself and in the other cerebral arteries no evidence of atheroma was obtained. A few small patches of atheroma were found in the coronaries, and in the aortic commissure and abdominal aorta there were one or two small plaques and in the femorals slight atheromatous changes, but the radials were neither thickened nor tortuous. The heart was not hypertrophied but the kidneys showed fine granular changes and a few cysts. The lungs, liver and kidneys were greatly congested. The contents of the small and large intestines were blood-stained. This blood had escaped from a sub-mucosal point in the first part of the duodenum.

A case of aneurysm of the left posterior cerebellar branch of the basilar artery which at autopsy was found to have ruptured and given rise to a mass of blood-clot which compressed the pons, cerebellum, medulla, and the occipital lobe of the left cerebral hemisphere in a married woman, aged 42, *who, when aged* 25, *and eight months pregnant of her second child, had suffered a transient hemiplegia affecting the right face, tongue, arm and leg, and who for five months before death had complained of slight epileptiform (petit mal) attacks with visual sensations. Nineteen days before death this patient had a seizure, followed by a second twelve days and a third ten days before the fatal issue. On admission to hospital, delirium, paresis left face and right hemiplegia of arm and leg, tenderness in left occipital region and stiffness of the upper part of the neck, left cerebellar posture of head, no nystagmus, early optic neuritic changes right.*

Case 22 (43124/1909).—Anne S., aged 42, married, was admitted to the London Hospital under the care of Dr. F. J. Smith, on November 19, 1909, and died on November 24. She married in 1885, and had had two children and no miscarriages. No history suggesting primary or secondary syphilis could be obtained. In 1889, at the age of 22, left inguinal colotomy for "stricture and ulceration" of the rectum was performed at St. Mark's Hospital. This stricture was thought to have had syphilitic origin. At the age of 25, when eight months pregnant of her second child, she suddenly woke one morning to find the right half of her body—face, tongue, arm and leg—paralysed and some difficulty with speech. The face and speech rapidly improved, and before her baby, which is still alive and now a healthy young woman, was born, the weakness of the extremities had largely disappeared. During the years 1892 to 1908 her health was good.

Between June and November, 1909, the patient had suffered five or six attacks of "blindness lasting a few minutes." In these attacks she never lost consciousness, and after ten minutes or so seemed perfectly well. The attacks were not accompanied by any headache, and in them she never vomited.

On November 5, 1909, nineteen days before death, she was seized with a sudden severe pain in the head and a sensation as of a "pulling of the back of her head as if her head were in a vice." This severe pain in the occipital region was accompanied by "aches all over." After the attack the left half of her face became "funny and weak." On November 12, she suffered from another "turn" in which she vomited once and became very "queer" and "quiet." On November 14, she suffered a second seizure "exactly like that on the 5th." The headache at this time was severe and her neck became "fearfully stiff and tender." After the headache ceased she was drowsy and suffered from "fever." With these attacks no weakness of the extremities was noticed.

On admission, the patient was an obese woman, weighing 13 st. 2 lb., who looked old for her years. She lay in bed moaning and groaning, and absolutely helpless. Shortly after admission delirium with outbursts of loud, frequent shouts of foul and filthy language set in. She mistook the people around her for her own relatives, and abused them. She was suspicious, but at times would listen to reason, and could with persuasion be got to answer questions. On command, simple orders were carried out. She complained of pain and tenderness over the region of the left occiput and nape of the neck, and greatly resented all attempts at turning her head. The head was held strongly flexed with the chin pointing towards the left. Under observation, she did not vomit, nor did she exhibit any irregular, involuntary movements.

The right arm and leg were spastic and paretic. All movements on this half of the body were slowly carried out and clumsily performed. No sensory loss was discovered. The right knee-jerk was exaggerated, the left could not be obtained. Ankle-clonus was not elicited. On the right side the plantar response was extensor, on the left flexor. The arm-jerks on the right side were brisk, on the left only doubtfully obtained.

The left fundus appeared healthy. On the right side the veins were engorged, the arteries unaffected, and the edges of the pink disc indistinct and blurred. Hearing was not affected.

Ocular movements were natural. Ptosis and nystagmus were absent. The pupils were small, regular in outline, and reacted definitely, though sluggishly, to light. The left face was paretic in its lower half, but moved fairly in emotion. The tongue could be protruded straight and held steadily. The sphincters were under control.

The rate of the pulse varied between 96 and 120. The temperature was raised to 103° F. Respirations were shallow—36 per minute. The tongue was thickly furred. The chest moved naturally. The heart was not enlarged and the cardiac sounds were natural. In the left inguinal region was an old colotomy wound, with healthy appearance. The liver reached below the costal margin, and its edges could just be felt. The urine contained neither albumin nor sugar.

On the nights of the 20th and 21st the patient became wildly delirious and excited. Next morning she was comatose, and the right side of the face was seen to be paretic. After this the pulse began to fail, and became variable in force and rate, tending to about 88 per minute. On the 22nd she could be roused with difficulty, cried out repeatedly, understood apparently some commands, and at times uttered a feeble unintelligible reply. Death occurred with increasing coma, grouped rhythmic respirations, flushing and cyanosis of the face and hyperpyrexia (T. = 105·6° F.) on the morning of the 24th.

At the autopsy (P.M. 1166/1909), performed on November 25, a ruptured aneurysm on the left posterior cerebellar branch of the basilar artery was found. The middle peduncles of the cerebellum, the pons, and the medulla oblongata were compressed on the left side by a lump of firm, brown-pigmented clot, and the meninges around this mass of clot were deeply stained, and of a red-brown colour. There was no softening of the brain, which weighed 1,134 grm. No evidence of atheroma was found either in the aneurysm or in the other cerebral arteries, but in the arteries of the general circulation moderate atheromatous changes were seen as fatty and calcareous plaques scattered here and there over the aorta, coronaries, &c. The heart weighed 361 grm. The lungs showed severe acid digestion and acute purulent bronchitis of the lower lobes. Two and a half inches above the anal orifice was an impervious stricture of the rectum, for the relief of which a permanent colostomy had been made; below this opening the mucous membrane of the colon and rectum was contracted and atrophied.

A case in which the partial rupture of an aneurysm at the junction of the left anterior cerebral and anterior communicating arteries occurred suddenly in man, aged 53, whilst watching a football match. Attacks of drowsiness and delirium, epileptiform seizures, clumsiness in movements, left frontal headache, left optic neuritis. Death on twenty-seventh day.

Case 16.—30191/1911: Joseph M., aged 53, married, was admitted to the London Hospital under the care of Dr. Francis Warner on January 26, and died on February 4, 1911. He had followed the trade of carpenter for thirty-five years, and until the onset of the illness from which he died had enjoyed good health. He married at the age of 26, was a widower, and by his wife had had five children, four of whom survived, aged respectively 25, 23, 21, and 19 years. He denied venereal infection and exposure. In early life he had had attacks of "typhoid" and "typhus" fevers; in recent years he had suffered on three occasions from "influenzal colds with fever and pains in the back."

During the latter part of December, 1910, and the first days of January, 1911, he had been feeling particularly well. On January 7 he went to watch a football match, and, whilst looking on, "came over queer, shivered, and developed severe frontal headache." He was taken home in a cab and put to bed. Consciousness was not lost; vomiting set in some hours later on the same day; he vomited several times, and brought up large quantities on each occasion. With the headache and vomiting he himself became irritable and ill-tempered. The pains in the head were chiefly vertical and frontal. He remained in bed at home from the 7th until the time of admission, complaining of a persistent headache of varying intensity; whilst in bed at home he vomited on several occasions, and was very constipated. Suddenly on the night of the 12th he became noisy, talkative, and delirious, saw black objects before his eyes, and made attempts as if to seize them. Next morning he was better again, and seemed "his normal self." A few days later, however, he began to "talk strange, and behave as if not right in his mind," but at the same time slept well. On January 19 he suffered from his first "fit"; in it he threw himself about, opened and shut his mouth repeatedly, and turned his eyes upwards. On the nights of the 20th and 21st he had three such attacks, "lasting on him altogether two hours and a half." On admission his daughter said that he had not recognized anybody since the 22nd, and had passed his urine and motions into the bed since the 24th. A letter from his doctor stated that albumen had been present in the urine on the 7th.

On admission, the patient was a well-preserved man, aged 53, of healthy appearance. He was dazed, drowsy, and lethargic. In answer to questions, he usually gave some reply after a pause, but the reply was pointless and irrelevant, rather than incorrect. He could give no connected account of himself, and when asked who he was, where he lived, or how long he had been ill, merely uttered some incomprehensible sounds. Apparently hearing was not affected. Attention after he had been roused was fleeting. He took no interest in his surroundings, and in general lay vegetable-like. Spontaneous movements of the hands were occasionally seen, but of the face and legs rarely. He showed evidence of recent wasting, was dusky and cyanosed; his face was expressionless and drawn. The mouth usually remained half-opened, exposing an extremely harsh brown tongue. The lips were dry and the pharynx filled with mucus. The fairly preserved teeth were covered with sordes and dried secretion. The skin was harsh and dry, and œdema was not present. Large varicose veins coursed over both lower extremities, and pigmentary scars, apparently secondary to old and healed varicose ulcers, were seen on the front of the shins. No suggestion of syphilis was discovered.

The apex-beat of the heart was felt in the fifth interspace, a quarter of an inch outside the mid-clavicular line. The area of superficial cardiac dulness was small. The clavicles were raised, the sternum fixed, the chest barrelled, and the respiratory movements small. No abnormal dulness was found over the lungs. The breath sounds were feeble and distant, and expiration was prolonged. No abnormal cardiac bruits were heard. The rate of the pulse was 96 per minute; before death it fell to 66. The temperature was slightly raised, to 99·5° or 101° F. throughout the illness. The superficial arteries were tortuous and felt thickened. The blood-pressure, as measured by the Riva-Rocci apparatus in the right brachial artery, was 180 mm.

The liver was not enlarged and its edge could not be felt. The splenic dulness was not large and the lower pole was not palpable. No abnormal signs were found in the abdomen. The urine contained albumin and a thick cloud was obtained on boiling. The specific gravity varied from 1028 to 1030. On standing a thick deposit of amorphous urates was obtained. Sugar was not present.

On the three days preceding death, February 1, 2, and 3, the patient exhibited several epileptiform attacks; the movements chiefly affected the left upper extremity and were followed by loss of consciousness, dilatation of the pupils, and the disappearance of the corneal reflex. Each attack lasted from a half to two minutes and was accompanied by relaxation of the sphincters. After the attacks he sank into a quiet and drowsy state. Headache was a constant complaint and was referred to the left temple and above the left orbit. As an in-patient he did not vomit.

There was no absolute paralysis of any limb. Irregular, inco-ordinate, clumsy movements of both hands occurred from time to time. Spasticity was not present and the tone of the muscles was little altered. Sensation was difficult to test, but no loss could be determined. Dermatographia was easily elicited.

The knee-jerks were exaggerated, the response on the right side was somewhat brisker than on the left. Ankle-clonus was not obtained. The arm-jerks were brisk. On both sides the plantar reflexes gave a flexor response. The abdominal and cremasteric reflexes were just obtained. The left corneal reflex was readily elicited, the right only doubtfully obtained.

The right optic disc and fundus appeared pale, the veins were full, but the arteries small and the edges of the disc sharp. On the left side the disc was swollen and its edges blurred; the veins on this side were larger than on the right but the arteries appeared fairly normal. The drums of the ears appeared healthy.

Ptosis and nystagmus were absent and ocular movements free. The pupils were equal and reacted sluggishly to light. Facial movements were rarely seen, but no paralysis could be made out. The movements of the tongue, larynx and palate were unaffected.

The patient suffered from retention of urine and dribbling overflow incontinence. He was constipated and the bowels were only opened with enemata. Death occurred from respiratory failure on February 4, twenty-seven days after the first seizure.

At the autopsy (P.M. 104/1911) performed on February 4, a ruptured cerebral aneurysm, of the size of a pea, was found at the junction of the left anterior cerebral with the communicating arteries. Around the aneurysm was much hæmorrhagic matting of the meninges. All the cerebral ventricles were filled with blood-clot which had found an entrance through into the left lateral ventricle by local softening and erosion. At the base of the brain was much intradural and intraarachnoid hæmorrhage. The brain weighed 1,535 grm. There was no visible atheroma of the cerebral arteries, but slight atheroma was seen elsewhere, a few fat-flecks in the coronaries, and pin-head fat-flecks in the commissure and descending aorta. The weight of the heart was 290 grm. The lungs were emphysematous and showed a fibro-calcareous nodule at the apex of the left lung and a few smaller nodules in the right upper lobe. The other organs appeared healthy.

On microscopic examination the aneurysm was found to have a thin wall made up of fibrous tissue with some slight fusiform thickenings. Some of these thickenings appeared to be fibrosed muscularis, others fibrotic thickenings of the intima in which a few elastic fibres were seen.

A case in which the rupture of an aneurysm of the left anterior cerebellar artery led six days later to the death of a young man, aged 22. Drowsiness, left facial paralysis, bilateral corticospinal interference, left cerebellar posture of head, stiffness of neck and blood in the cerebrospinal fluid.

Case 29 (32669/1909).—Arthur P., aged 22, stable-boy, married, was admitted to the London Hospital, under the care of Dr. Leyton, on November 24, 1909, and died on November 25.

His father stated that his health had been good until November 19, 1909. In 1903 he had been laid up in bed at home for six weeks with "rheumatic fever," and in 1907 he suffered an attack of "gonorrhœa." On November 19, while in the stable he fainted, but after the faint did a full day's work, going a complete round with the cart. On the 20th he refused his food, said he "felt queer" but made no specific complaints. On the 21st he got up but was "half-asleep, talked to himself, and would not listen to what was said." On the 22nd and on the morning of the 23rd he seemed better, but about 11 a.m. on the latter day he had a seizure. At the time he was walking about the house, when suddenly he reeled, came over stupid and deaf. He was put to bed and when found at 5 p.m. was deeply unconscious. A doctor was called and shortly after midnight he was admitted to hospital.

On admission, he was semi-conscious and in general lay quiet in bed on his left side. He resented all interference and when spoken to made some unintelligible replies, continuing thereafter to mumble to himself. His face was flushed, his breathing stertorous. On the bridge of the nose was a scar obtained in his fall on the 19th. The right side of the heart was dilated but there was no evidence of any enlargement of the left ventricle. At the apex a systolic murmur was audible. With the exception of a few rhonchi, no abnormal signs were discovered over the lungs. The urine contained a trace of albumen but no sugar and no casts. The temperature varied between 101° and 102° F., the rate of the pulse from 92 to 116 and of respirations from 32 to 38 per minute. The blood-pressure in the brachial artery measured 110 mm.

The optic discs and fundi appeared healthy. All the ocular movements and the movements of the pupils were unaffected, but the patient could not be persuaded to fix his gaze upon any object. The left side of the face both in its upper and lower halves was paralysed. The tongue could not be protruded.

Both arms were held stiffly, the right more so than the left. The left arm from time to time twitched in an irregular manner. The right leg was paretic and the tone in its muscles increased. There was no rigidity of the muscles of the neck but the head was held flexed towards the left shoulder and the chin pointed towards the right. Sensation could not be tested.

The knee-jerks on both sides were exaggerated, ankle-clonus was elicited from both ankles, and the response from the plantar reflex on each side was extensor. Both wrist- and elbow-jerks were brisk. The abdominal reflexes could not be obtained. Urine and fæces were passed incontinently.

Consciousness was never regained. On the 25th the eye-movements were more dissociated and irregular and the head more definitely rotated to the left; true nystagmus, however, was not obtained. Intense photophobia was present. The reflexes varied, at times ankle-clonus was readily obtained, at other times this sign was absent. On the 25th lumbar puncture yielded clear fluid under pressure which contained a few red cells, excess of polynuclear cells, but no organisms. Death occurred six days after the initial "faint" on November 25 from respiratory failure.

At the autopsy (P.M. 1166/1909), a ruptured aneurysm of the size of a pea was found on the left anterior inferior cerebellar artery. All the other cerebral vessels appeared healthy, there was no cardio-vascular hypertrophy and all the organs except the lungs, which showed broncho-pneumonic changes, appeared healthy.

(B) EMBOLIC ANEURYSMS.

A case of infected endocarditis in a child aged 7 years, in whom, after a period of slight malaise, death occurred from cerebral hæmorrhage seven hours after a seizure consequent upon the rupture of an embolic aneurysm on the right middle cerebral artery.

Case I.—20861/08, Bessie G., aged 7, was admitted to the London Hospital under the care of Mr. Percy Dean on April 7, 1908, and died seven hours after admission.

The mother stated that the child had been ailing for some days but had never been really ill until the day of admission. On this day, whilst playing on the stairs, she suddenly fell down four steps and struck her forehead against the stairs. Immediately after the accident she was brought up to the hospital and admitted.

The patient was a well-developed child of 7. On admission she was unconscious. At intervals she showed irregular movements of the right upper and lower extremities; in these attacks the eyes were observed to roll slowly outwards, first to the left and then to the right, and the mouth was seen to twitch in an irregular fashion. The left upper and lower extremities were paralysed and stiff.

Both knee-jerks were brisk. On the left side the plantar reflex gave an extensor response, on the right a flexor.

The area of cardiac dulness was increased to the right of the sternum, but the apex-beat was not displaced either outwards or downwards. At the apex the first cardiac sound was replaced by a long, blowing, systolic murmur which was well heard in the axilla. The pulmonary second sound was accentuated. No abnormal physical signs were discovered in the lungs and no signs of any injury were present. The rate of the pulse was 68 per minute and of respiration 27 per minute, and the temperature was 96° F.

Chloral was administered to the patient by the mouth, but five hours later respiration began to fail. Artificial respiration was carried on for half an hour, at the end of which time the pulse could no longer be felt at the wrist.

At the autopsy performed on April 8, 1908 (P.M. 345/1908), a ruptured aneurysm of the size of two peas was found on the right middle cerebral artery about 1½ in. from its origin. The posterior part of the basal ganglia and the internal capsule had been almost completely destroyed and blood-clot was found in both lateral and in the third and fourth ventricles, whilst the convexity of the right cerebral hemisphere was covered with sub-arachnoid hæmorrhage. The heart, which weighed 113 grm., showed the appearances of progressive endocarditis, with large fibrinous vegetations on the mitral and aortic valves. The spleen was septic and the organs engorged. No other aneurysms or infarcts were discovered. Microscopic examination showed that the aneurysm was filled with organizing blood-clot and that the aneurysmal wall exhibited an area of necrosis with much round-celled and purulent infiltration around the artery.

A case of infected endocarditis with multiple infarctions and the formation of embolic aneurysmal dilatations on the left intracranial carotid, the left renal and the cœliac axis arteries in a girl aged 15. *Onset of acute manifestations with a seizure followed by aphasia and right hemiplegia thirty-two days before death: cardiac affection first recognized seven days before death, which occurred in consequence of bleeding into the lesser sac of the peritoneum.*

Case IX.—40136/1911, R.H.—Mary N., aged 15, was admitted to the London Hospital under the care of Dr. Robert Hutchison, on January 15, 1911, and died on February 15.

As a baby the patient had suffered only from attacks of whooping-cough and measles. She had never suffered from acute rheumatism or scarlet fever and had had good health until the onset of the illness for which she was admitted to hospital. About four weeks before admission, her mother stated, she had had a blow upon the head. This blow was not followed by untoward symptoms, but from it her mother dated the present illness. On January 8, 1911, the patient began to complain of indefinite aches and pains in her arms, legs and back; they were, however, slight, and she was not confined to bed. On January 14, the day before her admission, she had a seizure. She fell down, clenched her teeth and voided urine and fæces. The seizure was followed by a period of restlessness marked chiefly by irregular movements of the left arm and left leg. After the seizure until the time of her admission she never spoke.

The patient was a fairly well developed Hebrew girl of 15, with dark hair. She was not anæmic and the lymphatic glands were not enlarged. She was drowsy, irritable, and only moved when stimulated or in response to loud noises. No wounds were discovered on her head or body. The cheeks were flushed, more especially on the side of the face on which she had been lying. Erythematous patches were seen at all points of pressure.

The right arm and right leg were paralysed; the right arm was stiff, the right leg flaccid. High-grade movements of a choreio-athetotic type of the left hand occurred from time to time. The left leg was moved freely, apparently under volition.

The right knee-jerk was exaggerated and knee-clonus was obtained on this side; on the left side the knee-jerk was readily obtained, but was not exaggerated. No ankle-clonus could be obtained. On the right side the plantar reflex gave an extensor response, on the left a flexor. On the right side no abdominal reflexes could be obtained, on the left they were brisk. The elbow-jerk and wrist-jerk on the left side were normal, on the right these could not be elicited. The jaw-jerk was normal.

Sensation could not be tested. The right arm was blue and cold, the left of normal appearance.

The right optic disc appeared healthy, but the edges of the left were blurred and the whole fundus on this side had a ground-glass appearance. The drums of both ears appeared healthy.

Ocular movements were well carried out. The right eyeball appeared more sunken than the left. The pupils were equal, of moderate size and reacted briskly to light. The lower half of the right side of the face and the levator palpebraris on this side were paretic. The tongue could not be protruded. The head was rotated slightly to the left and the sternomastoid muscle on the right side appeared to be completely paralysed.

The patient was constipated and the bowels only moved after the administration of enemata. Urine was passed incontinently.

On admission the rate of the pulse was 120 per minute; the beats were regular and equal. The apex-beat of the heart was seen and felt in the fifth space just inside the mid-clavicular line. At all the areas both cardiac sounds were heard and no murmurs were present. The pulmonary second sound was slightly accentuated. The rate of respiration was 24 per minute. The movements of the chest were unaffected and the breath sounds were unaltered. The abdomen was somewhat sunken. Neither the liver nor the spleen could be felt, and the urine contained neither albumen nor sugar. The lips were dry and the tongue coated with a brownish fur. The tonsils were large.

The temperature on admission was 100° F. The temperature for eight days after admission was raised and somewhat irregularly hectic, ranging between 101° in the evenings and 99° in the mornings. On the three days before death after an apyrexial period, the evening temperature rose, and on the evening of February 13 reached 102° F.

On January 16 lumbar puncture was performed and 30 c.c. of cerebrospinal fluid were withdrawn. This fluid contained many red blood corpuscles, a few lymphocytes and a large excess of polynuclear cells. On January 26 lumbar puncture was again performed; the fluid on this date was under pressure, but on centrifugalization it yielded no cells and cultures made from it proved sterile.

Ten days after admission the patient began to talk again, and the right hemiplegic weakness commenced to clear, and until about February 6 the patient seemed to be making a good recovery; on this day the patient vomited and the first cardiac sound became much weaker. On February 8 a loud mitral systolic murmur was heard for the first time. After this date the patient became more ill, wasted, refused food and gradually sank, apparently from exhaustion and increasing anæmia. Death occurred on February 15, thirty-two days after the initial seizure.

At the autopsy performed on February 15 (P.M. 137/1911), an aneurysm, 3 cm. in diameter, partially walled with blood-clot, was found on the cœliac axis artery. This aneurysm had ruptured into the lesser peritoneal sac. The peritoneal cavity contained 595 grm. of blood-clot and 1,136 c.c. of serous fluid. The heart, which weighed 141 grm., showed a condition of ulcerative endocarditis, there being one large slimy, polypoid and many small granular vegetations on the mitral valve. In the hilum of the left kidney the left renal artery was found dilated and thrombosed; this thrombosis had given rise to a total infarction of this kidney. The right kidney showed a condition of acute hæmorrhagic nephritis. The spleen was septic and weighed 169 grm., and the liver showed much parenchymatous degeneration.

At the point of bifurcation of the left intracranial carotid artery was a thrombosed aneurysmal dilatation caused by a septic embolus. The left basal nuclei and the adjacent part of the left centrum semi-ovale showed white and yellow softening, and above the posterior limb of the fissure of Sylvius was an area of central softening measuring 3 cm. in diameter.

John Bale, Sons & Danielsson, Ltd., Printers, 83-91, Great Titchfield Street, London, W.

WELLCOME INSTITUTE LIBRARY